Personal Dr. Zafar Ashraf M.D. Pharmacology Deptt

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EDITORIALS:		
-Medical Journalism in 20th century	1	G.M. Malik
-From GIANTS to ROBOTS.	3	M.A. Jalu
Guest Article:		
-Current concept on the management of Gall stones	7	R. Tandon
CLINICAL REVIEW:		
-Cardiac Tymponade	10	P.K. Shah
Leading Articles:		
-Coronary Artery Bypass Grafting VS PTCA	19	P. JAIN, U. Koul
-NIMO DIPINE for Treatment of Stroke	23	S.A. Tanki, Y.A. Quazi, Z. Jeelani
New Horizons in Surgery :		
-Laparoscopic Cholecystectomy	28	N.A Wani,T. Fazili, U. Shah
-Intra-ocular lens Implantation	31	S.J. Ahmed.
Origional Articles:		
-Hearing Impairment in School childern of Kashmir (Epidemiological profile)	34	Z. Ahmed, S.A. Ahad
-ACTIVATE clotting Time Estimation in Heparin reversal during open heart Surgery	37	A.G. Ahanger, Mehrajdin
-Mixed germ cell Tumors of Testes	40	Chowdhery N.D., A.R Khan
Case Reports:		
-Papillon-Lefevre Syndrome	43	M. Quazi, S. Manzoor
-Spinal Hydated cyst causing cord compression	44	G.R. Mir
-PNEUMOENCEPHALUS following Nasal polypectomy	47	N.A. Bhat
PRACTITIONERS COLUMN:		
-AIDS (Part II)	49	G.H. Hajini
-Haematuria, Do we manage it correctly!	53	G. Jeelani, Mumtaz D.
CME Programme:-		
-Seminar on T.B in Valley (How we face it!)	56	M. Yousf
Students Page:		
-Scenario of T.B. in SAARC Region	60	
Meet the Professor:		
-Prof. S.N. Ahmed Shah	61	
Foundation of Medical College Kashmir	63	P. Chesti
In Memorium:		
-Farooq Ashai	66	G.H. Hajini, G.R. Mir
-A.A. Beigh	67	Margoob, Shah, M. Yousf
Overseas diary:		
-History & Training of foreign Medical graduates in U.S.A	69	Farooque A. Khan
Communications to the editor:	71	

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-> Meet the Professor.
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JK Practitioner 1996; 3:1 1-2

Editorials

MEDICAL JOURNALISM IN 20TH CENTURY

"PUBLISH OR PERISH"

John Ziman, quoted "The object of science is publication". Science started formally in Britain within the Royal Society in 1660. One of the Ist thing that the Royal Society was asked to do was to start a journal. Before that scientists used to be very secretive about their work and kept it to themselves. Thus the Ist publication of major scientific journals descavans and the philosophical transactions of the Royal Society started in 1660. The primary loyalty of Editors is to sciences and they should not dilute their standard of newness, trueness and importance.

Initially there was dominance by British journals which was followed by American and European journals. During 20th century there has been number of journals published from Asian, African and Australian countries but they are still facing dominance from west. The Ist Indian Medical Congress held 24-29 Dec. 1894 was published in B.M.J (16 Feb. 1895 at Calcutta). 50% of all papers published from 3rd world are from India yet India in 3rd world league stands 23rd in citation rate, behind countries like Jamaica, Thailand, Kenya, Uganda and many others. A team of British journal lead by Stephan lock Editor British Medical journal during his visit to India, found that there are enough of journal published in India, but maintained that hardly 1 to 2 journals are of international standard. One of the reasons being that every doctor prefers to send their best papers abroad rather than local journal. Presently there is lot of competition among the various international journals, and besides of western of Chauvinism e.g. American Journals have a paroma about to quote journals from England.

There is lot of difference among various editors in choosing articles for publications received from different parts of world. In 3rd world journals articles on infections disease will be preferred for publication whereas in west and Europe degenerative, and autoimmune disease get wide publication and acceptance. It is worth while to have ones own journals from every country or state preferably in International language e.g. English. The new England Journal of Medicine started as a local journal from Philadelphia (U.S.A), as the organ of Massachusetts Medical society and now has world wide readership.

During last few decades there has been criticism against the Editors, being selective in giving preference to some particular authors, articles depending upon their own discretions irrespective of standard of articles, which has stimulated many scientists to establish standard journals in their countries in field of Medical Sciences in English or even local languages e.g. Spanish, French, Russian, German, Arabic etc. In India there are more than 100 languages, but till todate every Medical journal is in English language. In India almost from every state especially South and Western regions good number of journals are published by various Medical Association, Societies or individual. In J&K State, it was November 12, 1994 that we started our Ist Medical Journal JK Practitioner with an international editorial board to review the articles and manuscripts. It has already published 5 issues (quarterly) till todate and has received lot of appreciation from here and abroad by our readers.

During last few years the medical Editors felt a need to have World Association of Medical Editors (WAME). The Ist meeting was held at Belgium, Italy on March 16, 1995 and discussed global problems concerned to medical editors. This body will train, encourage new editors in medical journalism besides have common network of communication all over the world with its members. Secondly in India, Association of Indian Medical Editors was established 3 years ago and held its 3rd conference at Lucknow on August 14-16, 1995 at Lucknow recently and decided to promote and improve Journal in India. We hope this attempt of various Associations of Medical Editors at National and International level will definitely improve medical journalism and bring 3rd world authors, at par in this field. With the West. Medical Journalism has given birth to hundreds and thousands of books, Monograms etc. all over the world. New trends like presenting Journals on CD ROM Diskettes & Audio-Visul Cassettes has started in West and is picking up fast. E-Mail, Fax and midline services had made it easier for reader to get articles of their choice from any Journal by sitting at home anywhere in world.

G. M. Malik MD, FACG
Chief Editor

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"The Student, practitioner requires at least those things to stimulate and maintain his education, a note book, a library and a quin-quennial brain dusting. Fora teacher and doctor, a good library is indispensable. For clinicians, Osler had a special admonition. To study the phenomenon of disease without books is to sail an uncharted sea"

"Sir William Oslar"

HAPPY NEW YEAR

GREETINGS TO OUR

READERS, SUBSCRIBERS

AND

CONTRIBUTORS.

This issue is dedicated in memory of Dr. A. A. Beigh (HoD Psychiatry Deptt.) who died in December 1995, on behalf of editorial and advisory board of JK Practioner as well as teachers association Medical College Srinagar and Jammu.

JK Practitioner 1996; 3:1 3-6

Editorials

FROM GIANTS TO ROBOTS

"The First Century of Neurosurgery"

The lack of understanding about the localisation of the brain's functions and consequently about the lesions in the brain, prevented surgeons from thinking of neurological surgery as an independent field, even after the advent of anaesthesia, antisepsis and asepsis in the second half of the 19th century. Later, neurologists could localise lesions in the central nervous system but, understandably, not many surgeons showed any interest in operating on the human brain because of the anticipated difficulties. Despite archeological and archival evidence that various procedures were carried out on the head many centuries age, the information that would provide a scientific basis for these procedures is lacking. At the end of the nineteenth century a British Surgeon, William MacEwan in Glasgow, was the first man in the world to operate on the brain and the spinal cord. Richman Godlee, William Bennett and Victor Horsley followed his lead and set the trend for surgery on the central nervous system. In 1884, Godlee resected a brain tumour, and in 1887 Horsley was the first person to remove a spinal cord tumour. He also attempted a neurotomy of the Gasserian ganglion on a patient with tic doloureux. The first posterior rhizotomy for pain was performed in 1888 by Bennett.

As the 19th century came to a close, more and more brain operations were reported, but not many surgeons were prepared to devote their full time and attention to this new field of surgery, mainly because of the very high operative mortality rate (greater than 50%). Many gave up, but those who persevered made a lasting impact and their names are written in the history books. One such surgeon was Harvey Cushing (1869-1939). Names such as Walter Dandy, Percival Bailey, Norman Dott, Sir Geoffrey Jefferson and Herbert Olivecrona became known and these men are now recognised as "Giants" in the art of neurosurgery.

At present, we are experiencing a phase of computer-tronics in neurosurgery with emphasis on their application to the minimally invasive procedures. These techniques are proving highly beneficial to precision and safety in neurosurgery. The American Association of Neurological Surgeons thought that the closing decade of the century which saw the start of this speciality was a fitting time to be designated the DECADE OF THE BRAIN. And thus it was so proclaimed by the President of the United States of America.

Revolutionary Landmarks

Let us now review the modalities that have had a major influence on bringing this speciality to the state of the art that it has reached today, and comment on the current trends.

Since 1950 every neurosurgeon who has operated on intracranial conditions can not but acknowledge the tremendous contribution of Greenwood for his invention of bipolar diathermy. The depth and narrowness of the operating field has always been a big challenge to the operating surgeon and improvements were, of necessity, continually being made to the source of illumination. The operating microscope was the solution to poor visibility, and in addition it provided a magnification of the working field.

Probably the single most influential tool in changing the whole concept of diagnosis and management of disease in general and neurological disease in particular, is computerised tomography. This superb British invention was first installed in Atkinson Morely Hospital, London, in 1971, having been produced by E.M.I Medical Ltd, under the direction of Hounsfield. 1981 was the year of the M.R.I., again from Britain, and this opened a new era for clear, crisp, and non-invasive imaging of normal and abnormal structures. M.R.I. almost completely eliminated the handicap of the previously meagre information that could be elicited for certain areas of the brain, for example the brain stem, and it also proved to be superior in spinal cord imaging. Digital angiography, S.P.E.C.T and P.E.T. scans are used widely for specific situations. Ventriculography, myelography, plain tomography, and at times plain skull X-rays, are being used less and less in present day neurosurgery. This reminds us of the lack of enthusiasm shown by Cushing when Dandy introduced ventriculography in 1918. Cushing believed that clinicians would come to rely more on the test than on clinical examination. This could well have happened, but in spite of his well known idiosyncrasies even Cushing would have appreciated this facility and thanked the people who were to make possible such wonderful neuroimaging. The list of excellent instrumentation and aids available in the operating theatre today is very long, but the ultrasonic surgical aspirator needs a special mention, as it makes resection of the tumour a far easier proposition. In addition, using this machine reduces the operating time and therefore the retraction of the normal brain, which in turn leads to a further reduction in morbidity. Early in the 1970's, more and more neurosurgeons started using the ultrasonic aspirator as well as the laser, but over the years use of the laser in neurosurgery has diminished whereas ultrasonic aspirators are used would-wide, mainly because they are highly efficient and very useful.

Quest for Excellence

Without doubt, the neurosurgical skills and the greatly reduced surgical morbidity and mortality known today, would not have been possible if the neurosurgeons had not received tremendous help from neuroimaging, improved instrumentation, and improved wuality of anaesthesia and intensive care. Surgeons in different parts of the world have worked devotedly to improve upon the foundation of the art and understanding of neurosurgery laid by people such as Cushing and Dandy.

By the sixth decade of this century, comprehensive physiological realities, clear and detailed pathology analyses, and classification of neoplastic and other conditions of the nervous system, were the bricks on which the surgeons would build. In the next decade we see ever higher standards of performance, and reports were published from different continents showing that "zero" operative mortality was being achieved in cases of benign intracranial lesions. A mortality of under 3% was reported in cases of intrscranial aneurysms (including the posteior circulation), and an even lower mortality was achieved in arteriovenous malformation. Whereas safer and more complete tumour removal and protection from the rupture of the vascular lesions was possible with very small or zero mortality rates, the same could not be said for operative morbidity, although this risk too had been reduced considerably. Using new methods and high technology (at high cost), safer forms of surgical treatment were developed in an endeavour to reduce morbidity, and these were received enthusiastically by the medical profession.

Interventional Radiology

Although initially greeted with scepticism, interventional radiology became an importment part of a tertiary care neurosurgical service within ten years. Like other surgery fields, neurosurgery also appreciated the fast growth of this form of treatment to the extent that some neurosurgeons elected to receive full training to practise this art. In the 1990's, it is considered the treatment of choice in the majority of cases of AVM and vascular fistulae, and as a preoperative first stage procedure for vascular tumours. Already aneurysms are being protected and stroke patients are being treated by this procedure. Not only has the use of interventional radiology led to a reduction in mortality and morbidity, it has also reduced the length of the patient's hospital stay, thereby reducing the cost of the treatment.

Stereotactic Surgery

Although a stereotactic frame was devised 100 years ago, its use for the localisation and treatment of lesions did not become popular until the 1950's. However, the intital enthusiams died down and it only revived about 15 years ago. Now, is use is almost universal. Many different frames have been developed by individual surgeons in various centres. The majority of these frames are compatible twith the C.T and M.R.I. machines, because their uses do in fact complement each other. The role of the stereotactic procedures in the verification of deep, inaccessible and small lesions, particularly in the eloquent areas of the brain, is well known, but recently more and more surgeons are using stereotaxy to plan craniotomies for different lesions as well as to deliver the medications in brachytherapy. Frameless stereotactic surgery is a recent development for the localisation of lesions with the help of computerized manipulation of the images. This goes along with the current technical developments, and in fact robots have been used to perform some intracranial procedures. All this of course requires very high-powered standards of computer technology.

Radiosurgery (knifeless surgery)

Radiosurgery involves the delivery of a high dose of ionizing radiation to a preselected target localised by the stereotactic frame. The "Gamma Knife" was invented by Lars Leksell in 1951 but the treatment of patients actually started in 1968 at the Karolinska Institute in Sweden. For many years it was used only by the team at that Institution, but towards the end of the eighth decade the Gamma Knife was being installed in other centres. In addition to its primary use in the treatment of vascular malformation, the Gamma Knife is being used in all kinds of benign brain and spinal cord lesions, as well as for functional neurosurgery. A similar machine is the linear accelerator; no doubt this has comparable scope in the treatment of various conditions, however, the superiority of the Gamma Knife, as far as neurosurgical practise is concerned, is now well recognised.

Minimally Invasive Surgery

Minimally invasive surgery would not have been possible without the improved methods, surgical aids, instrumentation and visibility that preceded its advent. Key hole surgery, a Japanese concept, found rather a limited scope, the same, however, cannot be said about endoscopic surgery whose application has increased since the beginning of the present decade. It has been particularly useful in the management of intraventricular lesions and the treatment of hydrocephalus. This technique will soon become an integral part of good neurosurgery service.

Head Trauma

It has been known for many years, and is still true of present day practise, that the ultimate outcoe of a primary brain injury does not depend on the place, the surgeon or the treatment that is given. What alters the outcome is the prevention and prompt treatment of secondary injury to the brain. This aspect has been widely researched, mostly with regard to elimination of the factors that would indicate a bad prognosis in a head injured patient. Huge campaigns to prevent the head injuries in the first place were eventually successful, but it is the prevention of onset of the secondary assault that has been responsible for the decline in the number of patients in a persistently vegetative state, a potential legacy from any neurosurgeon treating head injuries. The current expectation in severe head injuries is: those who survive the primary injury have a better functional result; this is a definite improvement on the outcome compared with that in the late 1970's.

The year 2000 and Beyond

As is true for any other speciality of medicine, the future is going to be better for neurosurgery. There is no question that high quality treatment demands the availability of large financial resources at either state or private level. One possible reason why neurosurgery developed more quickly and continues to expand in the United States is that, owing to the very large and strong private sector in American medicine, money is available to set up the apparatus to make the sophisticated treatments available to patients. Yet the majority of the world's population lives outside the Unites States, Japan and Europe. The people of Africa, China, India, Pakistan, Bangladesh, and some countries from the former Soviet Union and the Middle East, suffer from the same neurosurgical diseases, but with higher mortality and morbidity because these countries lack the financial means to provide optimal treatment. And so a great many more needy patients throughout the world may come to benefit from the advances achieved in the 20th century. Let this be our aspiration for the 21st century.

** M. Ashrah Jallu, Department of Neurosciences*
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Guest Article

"CURRENT CONCEPTS IN THE MANAGEMENT OF GALLSTONES"

*Rakesh Tandon

Introduction

Gallstones have been believed to occur predominantly in the affluent Western countries, but recent studies in American Indians as well as many other communities including North Indians have shown this impression to be incorrect. Indeed, Pima Indians have about the highest prevalence of gallstones in the world, 70% of their women-folk acquiring the disease by 25 years of age[1]. Two epidemiological studies performed in Northern India, one in Kashmir Valley[2] and the other in Delhi[3] have shown the prevalence in this region to range between 4 and 60%. The prevalence in South India is possibly much lower as suggested by an earlier study conducted in rail-road workers in which it was shown that gallstones occurred 7 times more commonly in North Indians as compared with South Indians[4].

Gallstone types and causes of their formation:

Gallstones are known to be of two types, depending on their cholesterol content. Those that have >70% of their dry weight as cholesterol are called cholesterol gallstones whereas those that have <20% of their dry weight as cholesterol are called pigment gallstones[5]. The predominant type in Northern India is cholesterol gallstones. Of 46 gallstones obtained from our patients, 28 (61%) satisfied the above mentioned criteria of cholesterol stones but an additional 9 (19.5%) patients were also considered as belonging to the same category because of the presence of cholesterol supersaturation of bile bearing them even though they had somewhat lower cholesterol content[6]. In fact, many workers now label stones with >50% cholesterol content as cholesterol gallstones[7].

Why is such a categorization necessary? It is because cholesterol gallstones may be dissolved by giving oral drugs but pigment gallstones are not[8]. The reason for the above difference lies in the pathogenesis of the two types of gallstones being different. Basically, a bile that is supersaturated with cholesterol will result in cholesterol gallstone formation and a bile that is supersaturated with unconjugated bilirubin may lead to pigment gallstones. There may in fact be combinations of the two and depending on the predominance of the precipitated component the stone may be treated as cholesterol or pigment gallstone.

Cholesterol gallstones are the outcome of secretion of an abnormal bile (lithogenic bile) by the liver[8]. Cholesterol is kept solubilised in the bile by phospholipids and bile salts in two physical forms, mixed micelles and phospholipid vesicles. Either an excess of cholesterol or a relative decrease in bile salt or phospholipids could lead to supersaturation of bile with cholesterol which in the presence of a nucleating agent may result in gallstone formation. The commonest cause of cholesterol supersaturation of bile is excessive secretion of biliary cholesterol by the liver cells but in some patients this is combined with a decreased biliary secretion of bile salts. In order to achieve cholesterol gallstone dissolution it is important to suppress cholesterol synthesis and/or cholesterol secretion and, if possible, to increase the molar percentage of bile salts in the bile. Both these can be achieved by administration of two bile salts, chenodeoxycholic acid (CDCA) and its B epimer, ursodeoxycholic acid (UDCA)[8]. Another group of drugs has become available recently; they block HMG CoA reductase, the enzyme catalyzing cholesterol synthesis and hence have the potential to decrease cholesterol supersaturation of bile. A representative of this group drug is pravastatin[9].

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*Department of Gastroenterology & Human Nutrition
All India Institute of Medical Sciences
New Delhi-110029.

Studies during the last 25 years have clarified many aspects of formation of gallstones, yet the knowledge in this regard is rather sketchy and superficial[10]. Two logical questions which remain unclear are: why supersaturation of bile with cholesterol or bilirubin occurs in some individuals in the absence of an identifiable reason and why only in a proportion of patients with such biochemical changes do gallstones form? Whilst the first question remains unanswered, a possible explanation for the second question may lie in the fact that several other factors including impaired gallbladder motility, deficiency of nucleation inhibiting proteins and an excess of nucleation promoting proteins work in concert with cholesterol supersaturation to produce gallstones[10].

Management

Based on the above understanding of gallstone formation, one can speculate that any medical intervention that lowers the cholesterol saturation of bile will likely reverse the lithogenic process and may result in dissolution of cholesterol gallstones. CDCA and UDCA do the same as mentioned above. A similar action could be expected in the case of pigment gallstones but unfortunately no drug is available till date that would lower the biliary concentration of bilirubin. An alternative to drug therapy is pulverisating the stones (lithotripsy) by shock waves applied extracorporeally. Unfortunately, the basic lithogenic process persists in the individual and hence the moment drug intervention is discontinued stones start reforming (recurrence). This is why the standard treatment remains surgical removal of gallbladder (cholecystectomy) which can be achieved either through conventional laparotomy approach or through laparoscopy. **Prevailing concepts which guide our choice and timing of therapy are given below:**

1. Only 10 to 15% of gallstones ever become symptomatic[11]. Hence asymptomatic gallstones may be left alone except in special circumstances such as astronauts or people posted in places lacking medical facility.

Table 1

Some important figures regarding lap chole as reported by the Royal College of Surgeons, Confidential Comparative Audit Secure, England.

	Conventional cholecystectomy	*laparoscopic Cholecystectomy*
No. of patients operated in U K during 1991.	3988	2131
Mortality	0.9%	0.16%
Respiratory 3.4% 1.2% complications	3.4%	1.2%
Intraoperative bleed	0.7%	1.3%*
Bile duct injury	0.18%	0.31%*

*More in laparoscopic than conventional

Table 2

Mechanism of action, doses, efficacy and side effects of CDCA and UDCA

Mode of cholesterol Desaturation of bile	***CDCA***	***UDCA***
Cholesterol secretion in bile	decreased	decreased
Cholesterol solubilisation	micelles	Micelles and phospholipid vesicles
Intestinal absorption of cholesterol	decreased	decreased
Effective Dose (mg/kg)	12.15	8-10
Efficacy	60%	70%
Side effects		
Diarrhoea	50%	rare
Rise in SGOT/SGPT	common	rare
Rise in serum LDL cholesterol.	10%	0

2. Removing diseased gallbladder appears to be the best treatment at the present as it ensures that there will be no reformation of gallstones, nor development of carcinoma of the gallbladder.

3. If however, cholecystectomy is performed for an abdominal pain/discomfort arising from an organ other than gallbladder the patient will continue to have the discomfort - the so-called "post-cholecystectomy syndrome"[12]. Thus it is necessary to analyze carefully the patients, complaints and ensure that a definite indication of gallbladder disease exists, e.g. acute cholecystitis or biliary colic. Upto 50% of patients undergoing cholecystectomy may remain dissatisfied[13].

4. Although the effect of cholecystectomy alone are imperceptible on the gut motility and functions, when cholecystectomy is combined with vagotomy often bile acid - mediated diarrhoea appears[14].

5. The natural history of chronic cholecystitis is not known. It may well be compatible with absolutely normal life span.

6. There does appear to be a slightly increased chance of developing right sided colonic cancer following cholecystectomy[14].

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CARDIAC TAMPONADE

PATHOPHYSIOLOGY, DIAGNOSIS AND MANAGEMENT

Prediman K. Shah *

Cardiac tamponade is a clinical syndrome that results from an increased intrapericardial pressure and leads to impaired cardiocirculatory function. Over the years, it has become apparent that the spectrum of cardiac tamponade is relatively wide, ranging from an asymptomatic elevation of intrapericardial pressure recognized during objective evaluation, to extreme hemodynamic compromise in the form of severe hypotension or electromechanical dissociation and many variations between these extremes. It is therefore useful to think of cardiac tamponade as a continum on a scale of hemodynamic embarrassment rather than an all or none phenomenon[41,47].

Etiology:

Cardiac tamponade (Table I) can result from any condition that leads to a pericardial effusion or intrapericardial collection of blood or clot. The most frequent causes of tamponade, however, include trauma, malignancy, uremia, and idiopathic pericarditis[19]. On rare occasions, cardiac tamponade may result from pneumopericardium[48]. Pneumopericardium has been reported as a complication of trauma, barotrauma during positive end-expiratory pressure administration, infection with gas-forming organisms, during Valsalva maneuver, and cocaine inhalation[48].

Pathophysiology:

The normal pericardial cavity is a potential space with 15 to 50 ml. of fluid that, based on its chemical composition, appears to be pre-dominantly an ultrafiltrate of blood. The average intrapericardial pressure is about 0 mm Hg or slightly subatmospheric. Like intrathoracic pressure, it becomes a little more negative during inspiration and a little less negative during expiration.

Table I. Common Causes of Cardiac Tamponade

1. Trauma (hemopericardium): blunt, penetrating
2. Malignancy (lung, breast)
3. Uremia (acute renal failure, chronic dialysis)
4. Idiopathic pericarditis
5. Viral pericarditis
6. Collagen vascular disease (lupus, rheumatoid arthritis)
7. Acute and chronic radiation pericarditis
8. Postmyocardial infarction (cardiac rupture, Dressler's syndrome)
9. Postcardiac surgery (hemopericardium, clot-tamponade, Postcardiotomy syndrome)
10. Diagnostic/therapeutic intracardiac procedure (perforation during cardiac catheterization, percutaneous transluminal coronacy angioplasty valvuloplasty, pacemaker insertion)
11. Ascending aortic dissection (hemopericardium)
12. Infective pericarditis: spetic, tubercular, fungal
13. Pneumopericardium.

In addition to the respiratory fluctuations, the intrapericardial pressure also fluctuates with the cardiac cycle, being slightly higher during diastole and slightly lower during systole. The pericardial pressure-volume relationship is such that rapid accumulation of fluid (such as following trauma or cardiac rupture) leads to a rapid rise in intrapericardial pressure, and cardiac tamponade can thus ensue with collection of only small volumes of fluid. On the other hand, with a more gradual accumulation of fluid, the pericardial tissue is able to stretch and increase in content by hypertrophy. The capacity of pericardial space is thereby increased, dampening any expected increases in pericardial pressure until a much larger volume of fluid accumulates and the elastic limits of the pericardial sac are overcome. Therefore, with rapidly accumulating fluid, tamponade may occur with as little as 100 to 200 ml, whereas with gradually accumulating effusions, tamponade may not occur even with 1000 to 1500 ml of fluid.

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* Director of inpatient Cardiology Division of Cardiology Cedar - Scnai Medical Center. Professor of Medicine, University of California Los Angles (USA)

The steepness of the pericardial pressure-volume relationship after the inflection point also indicates that even small increments or decrements in fluid volume would have disproportionately larger effects on the intrapericardial pressure. With a rise in intrapericardial pressure approaching the right heart filling pressure (right atrial pressure), the transmural filling pressures decrease, resulting in reduced right ventricular filling and, consequently, reduced right ventricular output. Reduced right ventricular output, in turn, reduces the left ventricular filling. Consequently, the left ventricular stroke volume is also reduced.

It has been demonstrated in experimental models of tamponade that the primary determinant of reduction in cardiac output is right heart compression, with compression of the atria contributing more than the compression of the ventricles[12,13]. In an elegant study, Ditchey et al showed that with sudden increase in intrapericardial pressure, right ventricular output falls first. Left ventricular output is maintained for several additional beats before it shows a decline, confirming the primary importance of reduce right ventricular output as a cause of reduced left ventricular output in tamponade[10]. Therefore, changes in systemic cardiac output are primarily passive and reflect compression of the right heart, diminished right ventricular stroke volume, and decreased pulmonary blood flow. The hemodynamic consequences of cardiac tamponade, thus, include a reduction in stroke volume and cardiac output and eventually a decline in arterial pressure.

In the early stages of cardiac tamponade, compensatory changes tend to minimize the deleterious consequences of reduced stroke volume in an attempt to preserve cardiac output, blood pressure, and reginal blood flow. These compensatory mechanisms include increased ventricular emptying and ejection fraction, tachycardia related to sympathoadrenal stimulation, increased peripheral vascular resistance (caused by sympathoadrenal activation as well as, perhaps, by a reduced release of atrial natriuretic peptide), renal conservation of salt and water (to maintain central intravascular volume and pressure[34]). These renal effects may be related to increase renal sympathetic tone, increase in renal vein pressure, or the reflex consequences of the unloading of high pressure baroreceptors[34].

In experimental models of tamponade, regional myocardial blood flow is generally well preserved as long as aortic pressure is well maintained[23]. However, perfusion to the acutely ischemic myocardium decreases during frank tamponade in direct proportion to the decrease in aortic pressure[31]. The blood flow to collateral dependent as well as normally perfused myocardium in experimental models of chronic coronary artery occlusion tends to vary according to the net coronary perfusion pressure[49]. Tamponade-induced hypotension appears to be the dominant factor in reducing regional myocardial blood flow in the presence of well developed coronary collaterals[49].

Clinical Manifestations:

Presenting symptoms of cardiac tamponade may be quite nonspecific and frequently are related to the underlying primary disorder. However, tamponade may present with dyspnea, fatigue, syncope, hypotension, or even shock. Chest pain due to associated pericarditis may also be a primary symptom. Since the severity of disease consists of a continuum along a spectrum, some patients with objective evidence of tamponade may have few symptoms in the very early phase of tamponade.

With regard to physical findings, the classic Beck's triad of low systemic arterial pressure, elevated central venous pressure, and a quiet heart on examination are relatively infrequent, particularly in medical patients whose tamponade develops more insidiously than in victims of trauma or those who have undergone cardiac rupture[3]. The hallmarks of physical findings in tamponade are elevated central venous pressure, relatively clear lungs, and the presence of pulsus paradoxus. Other nonspecific physical findings that may be present include tachycardia, tachypnea, and muffed heart sounds. In additions, some patients may have pericardial friction rub, fever, and other associated findings related to the cause of pericardial involvement.

Central Venous Pressure:

Central venous pressure is invariably elevated, although it may be difficult to detect in some patients with an unusually thick neck. Careful examination of the patient is essential for correct identification of this finding. Contrary to popular belief, the mean central venous pressure decreases furing uninterrupted inspiration i.e. Kussmaul sign (lack of decrease or actual increase in central venous pressure during uninterrupted inspiration) is generally absent unless there is an element of associated constriction, in which case Kussmaul sign is frequently present[43].

In certain patients with tamponade, wither in early stages or when associated with concomitant volume depletion, the central venous pressure may be normal (low-pressure tamponade). In such patients, the diagnosis of tamponade may require assistance from additional tests such as an echocardiogram.

Pulsus Paradoxus:

Pulsus paradoxus is said to be present when there is a greater than normal decline in arterial systolic pressure with inspiration. The term pulsus paradoxus was coined by Kussmual to describe the paradox of continuing presence of audible heart sounds. Palpable arterial pulse during inspiration is absent in a patient with tamponade, reflecting an extreme decline in inspiratory blood pressure[27]. In normal subjects, systolic arterial pressure rarely declines by more than 10 to 12 mm Hg during quiet inspiration. Thus, a decline greater than this has been considered to indicate pulsus paradoxus. However, the magnitude of inspiratory decline may also vary with the magnitude of expiratory arterial pressure[9]. Therefore, Curtiss et al have suggested that pulsus paradoxus be defined as an inspiratory decline of systolic pressure of more than 10% of expiratory systolic pressure[9]. In their series, this criterion accurately distinguished patients with suspected tamponade whose cardiac output improved by more than 20% after pericardiocentesis[9].

Table 2. **Bedside Measurement of Pulsus Paradoxus**

1. Inflate cuff of a conventional mercury sphygmomanometer to levels above expected systolic pressure (200-250 mm Hg).
2. Slowly deflate cuff while listening over the brachial artery for Korotkoff sounds.
3. Note the level of pressure at which Korotkoff sounds first appear (A). If there is pulsus paradoxus, Korotkoff sounds are heard initially only during the expiratory phase of respiration.
4. Continue slow deflation until Korotkoff sounds are beginning to be heard continuously during inspiration and expiration. Note this pressure (B).
5. Determine the differnce in pressures A and B, this is the degree of pulsus paradoxus.

Pulsus paradoxus may be estimated at the bedside with a sphygmomanometer (table 2). Disappearance of a palpable radial pulse during inspiration is almost always associated with paradox greater than 20 to 25 mmHg and severe tamponade. Intra-arterial pressure monitoring is the most precise way to measure pulsus paradoxus. Pulsus paradoxus, however, may be difficult to assess in patients with an irregular rhythm or atrioventricular disssociation, severely hypotensive patients, and those with severe tachypnea.

Mechanism of Pulsus Paradoxus:

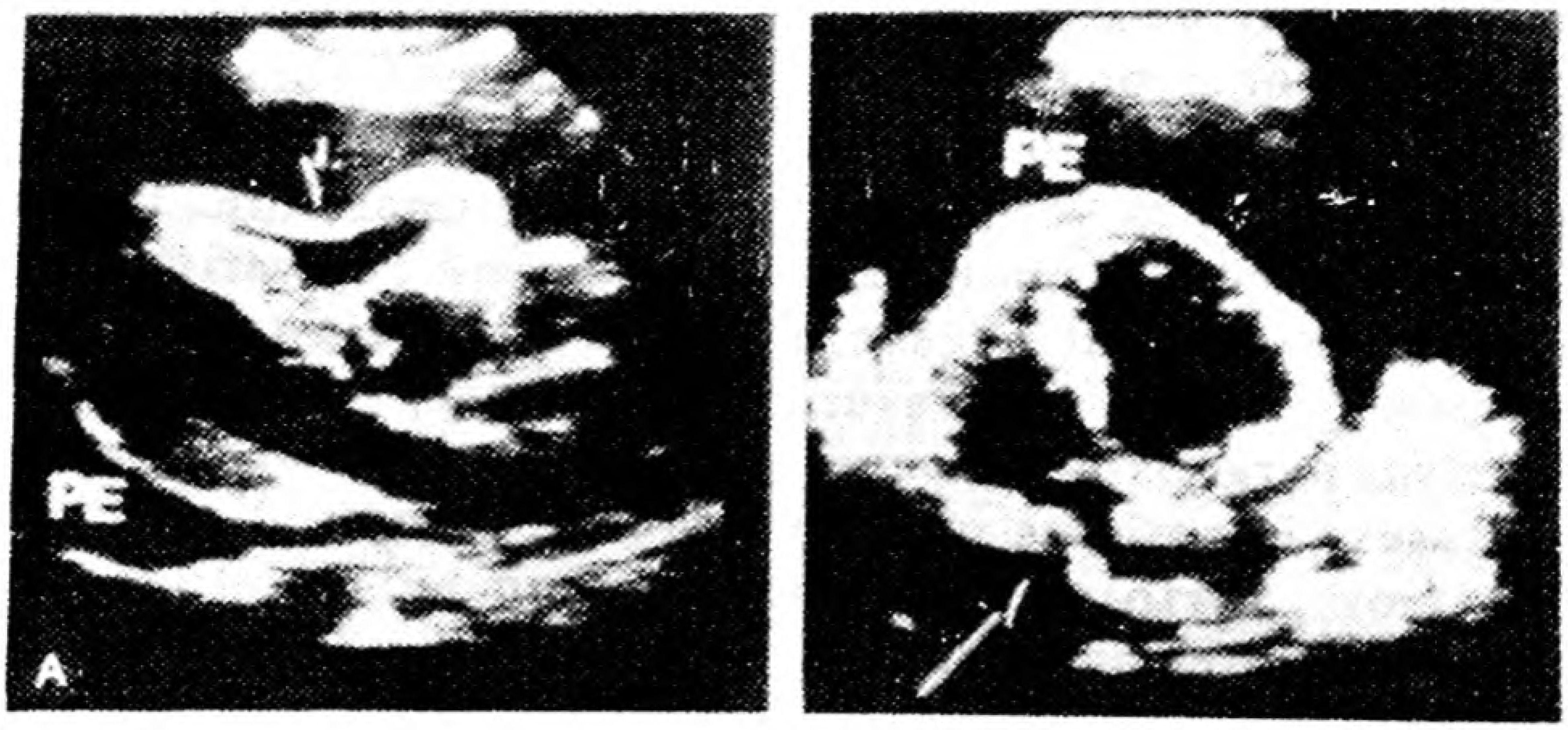
While many explanations have been offered to explain pulsus paradoxus, increased inspiratory inflow into the right ventricle and exaggerated inspiratory decline in left ventricular filling and output are key determinants[14]. In pulsus paradoxus, the minimal systolic and pulse pressure occurs at the onset of inspiration, whereas the maximum is noted at the beginning of expiration. The concept summarized by Reddy et al appears to be a satisfactory explanation that is in keeping with the key requirements[36]. With the onset of inspiration, there is a fall in intra-thoracic pressure that promotes increased vena caval inflow into the right heart. With the expansion of the pulmonary vascular bed, this increased flow is acxually accompanied by a transient decrease in the pulmonary venous pressure and a minimal decrease in systemic venous pressure. This favors right ventricular filling over left ventricular filling. In addition, the interventricular septum bulges into the left ventricle with a concavity toward the right ventricle because of a transient increase in right ventricular diastolic pressure compared with left ventricular diastolic pressure. Thus, left ventricular end diastolic volume and consequently the stroke volume decrease at the onset of inspiration.

At midinspiration, the increased venous return reaches the left side. Pulmonary venous pressure rises, increasing left ventricular filling. At this point, the left and right ventricular diastolic pressures are equal, and the septum returns to a neutral position. With onset of expiration, intrathoracic pressure rises, leading to a transient increase in pulmonary venous pressure above the systemic venous pressure. Left ventricular filling is thereby favored over right ventricular filling. Septum bulges into the right ventricle and right heart filling is reduced. Left ventricular stroke volume and arterial pressure increase with continues expiration and decreasing right heart inflow.

The left heart inflow eventually falls, and the septum returns to a neutral position with equal filling of the two ventricles. The arterial pressure returns to an intermediate level. Decreased left ventricular output during inspiration may also be due in part to increased afterload secondary to a reduction in intrathoracic pressure[32].

Pulsus paradoxus is not specific for cardiac tamponade since it may occur in other conditions as well (Table 3). Similarly, cardiac tamponade may exist without pulsus paradoxus in patients with preexisting elevations of left or right ventricular diastolic pressures[8, 35], with coexistent atrial septal defect,[50] or in severe aortic insufficiency. Absence of pulsus paradoxus during tamponade associated with an atrial septal defect may be due to constancy of right ventricular inflow during inspiration and expiration because of opposite effects of inspiration on vena caval inflow versus left-to-right atrial shunt. In aortic insufficiency, inspiratory decline in left ventricular filling from an aortic regurgitan jet, thereby eliminating one of the prerequisites for pulsus paradoxus.

Reddy and his colleagues have shown that tamponade may not be associated with pulsus paradoxus in uremic patients because of frequent preexisting elevations in left



ventricular end distolic pressure[35]. Pulsus paradoxus may also be absent when tamponade is more regional and when predominantly caused by a localized fluid collection or compression by a blood clot, as is common after cardiac surgery[21].

Table 3. **Tamponade and Pulsus Paradoxus**

Tamponade without pulsus paradoxus	
1.	Pre-existing elevations of left or right ventricular diastolic pressures.
2.	Coexistent atrial septal defect.
3.	Coexistent aortic insufficiency.
4.	Regional and asymmetric cardiac compression
Pulsus paradoxus without tamponade	
1.	Obstructive airway disease.
2.	Severe hypovolemic shock.
3.	Right ventricular infarction.
4.	Pulmonary embolism.
5.	Exposure to gravitational stress.

Diagnosis:

The diagnosis of cardiac tamponade should be clinically considered in patients with pericarditis or pericardial effusion and associated circulatory insufficiency, and in patients with circulatory compromise in the context of a disease process or circumstances likely to involve the pericardium. The presence of elevated central venous pressure and pulsus pradoxus are valuable clinical findigs of tamponade in these patients, but exceptions, as indicated previously, should also be considered. The chest x-ray is not directly helpful but may reveal indirect evidence of a pericardial effusion, e.g. globular heart or rapid enlargement of cardiac silhouette on serial films. Such findings, however, are not specific.

The electrocardiogram may offer valuable clues such as diffuse low voltage, especially when serial tracings show decreasing voltage indicative of a pericardial effusion. It should, however, be recognized that in the absence of serial tracings, isolated low-voltage electrocardiograms (EKGs) may not be particularly useful. Experimental studies have shown that changes in QRS voltage in pericardial effusion are not solely due to the amount of fluid but are significantly influenced by the electrical conductivity of the fluid as well[25]. In canine studies, Kramer et al showed that QRS voltage tended to decrease when saline-type fluid was introduced in the pericardium, whreas the presence of blood produced little change in surface QRS amplitude[25]. The EKG may also show nonspecific ST-T changes or PR segment and ST-T changes of acute pericarditis. Again, these findings do not necessarily indicate the presence of tamponade but are helpful in directing attention to pericardial involvement.

Electrical alternans, especially when it affects P-QRS, is a rare but quite specific finding in tamponade, whereas partial alternans (involving only QRS) can be observed with large effusions, usually but not always, in association with tamponade[17,46]. Electrical alternans is believed to result from pendular side to side motion of the heart when it is surrounded by a large amount of adhesion-free fluid. Electrical alternans may be most noticeable in one lead. At times, it may be necessary to record a Lewis lead toshow total alternans[46]. Since malgnant effusions are likely to be large and free of adhesions, it tends to be more common in these circumstances. Since electrical alternans may also occur in other clinical situations, notably during paroxysmal supraventricular tachyeardia, its diagnostic significance should be interpreted accorinf to the clinical context in which it occurs.

Echocardiography and Doppler Flow Studies:

Echocardiography has emerged as the single most important diagnostic tool for detecting the presence, location, and amount of pericardial effusion and for establishing the diagnosis of cardiac tamponade.

A number of echocardiographic findings have been proposed as indicators of cardiac tamponade. These include exaggerated parallel motion of anterior and posterior walls of the heart throughout the cardiac cycle (swinging heart); reciprocal respiratory variations in right versus left ventricular dimensions; abnormalities of mitral valve motion (peudoprolapse, abnormal inferior vena caval plethora with reduced inspiratory collapse, and monophasic [systolic] flow pattern); reciprocal changes in inspiratory flow across right - versus left-sided cardiac valves; collapse of right ventricular wall during early diastole; collapse of right atrial wall during late diastole lasting greater than one-third of the cardiac cycle; and collapse of the left atrium and the left ventricle in rare instances (Table 4, Fig. 1).[2,4-6,11,15,16,24,26,29,30,37,38]

Table 4. **Echocardiographic and Doppler Findings in Tamponade.**

*1.	Right ventricular collapse (early diastole).
*2.	Right atrial collapse (late diastole).
3.	Reciprocal respiratory variations in right ventricular and left ventricular diastolic chamber dimensions.
4.	Inferior vena caval plethora with reduced inspiratory collapse and monophasic (systolic) flow pattern.
5.	Swinging heart and mitral pseudoprolapse.
6.	Exaggerated inspiratory augmentation of flow-velocity integral across tricuspid and pulmonic valves with a reciprocal decrease across aortic and mitral valves.

* These findings are considered to be the most useful, sensitive, and specific for diagnosis of tamponade.

Echocardiographic-hemodynamic studies have shown that echocardiographic sing of tamponade may be detected at a stage when few or no clinical signs of tamponade exist, as well as at a stage when there is minimal impairment of cardiac output[26]. These findings confirm the current feelings that tamponade should not be considered an all or none phenomenon. Instead, it should be perceived as a continuum along a spectrum that ranges from echocardiographic findigs of tamponade with few clinical of hemodynamic manifestations to frank cardio vascular collapse.

Of all the echocardiographic findings, right ventricular and right atrial collapse are the most useful signs of tamponade. Right atrial collapse tends to occur earlier in the evolution of tamponade, and right ventricular collapse tends to occur somewhat later[16]. This temporal evolution is the most likely explanation for the purportedly higher sensitivity of atrial collapse compared with ventricular collapse and the higher specificity of ventricular compared with atrial collapse. Both findings are indicative of an elevated intrapericardial pressure that transiently exceeds intracavitary pressure at the time of collapse of the respective walls. Thus it is understandable that when right ventricular diastolic pressure or right atrial cavitary pressure is high secondary to associated diseases such as pulmonary hypertension, right ventricular hypertrophy, or tricuspid regurgitation, these echocardiographic signs may be abset, whereas reduced intracavity pressures with hypovolemia may allow these signs to appear with smaller increase in pericardial pressure. Leimguber et al have shown that right ventricular diastolic collapse tends to occur at a time when there is a 21% reduction in cardiac output[29]. Withdrawal of fluid in patients with tamponade shows that cardiac output improves first when right ventricular diastolic collapse disappears and further improvement occurs when right atrial collapse disappears[5].

Hemodynamic Findings

The hemodynamic findings of cardiac tamponade include an elevated right atrial pressure with amputation of the Y descent and preservation of X decent (consistent with predominantly systolic inflow into the right atrium), absence of Kussmaul sign, equalization (within 2 to 3 mm) of right atrial and pulmonary capillary wedge pressure (right and left ventricular end-diastolic pressures), and the presence of pulsus paradoxus (Table 5).

Table 5. Hemodynamic Findings in Cardiac Tamponade.

1. Elevated right atrial and right ventricular and diastolic pressures.
2. Equilibration of right atrial and right ventricular enddiastolic pressure with pericardial pressure, left atrial, left ventricular end-diastolic and pulmonary capullary wedge pressure.
3. Amputation of right atrial Y descent with preservation of X decent.
4. Absence of Kussmaul sign.
5. Absence of "dip and plateau" in ventricular pressure wave form.
6. Pulsus paradoxus in:
    systemic arterial pressure,
    pulmonary arterial pressure
    (out of phase with systemic).

In tamponade associated with pre-existing left ventricular failure, especially in volume over-loaded uremic and dialysis patients, left ventricular end-diastolic pressure may far exceed right ventricular end-diastolic pressure and pulsus paradoxus may be absent. The intrapericardial and right atrial pressure are, however, identical in all such cases of tamponade[35].

Subacute Effusive-Constrictive Pericarditis:

This clinical syndrome represents the consequences of combined tamponade and visceral pericardial constriction, which can result from radiation, malignancy, tuberculosis, and uremia. In constrast to patients with pure tamponade, these patients may have a positive Kussmaul sign along with a pulsus paradoxus. Following removal of pericardial fluid and relief of tamponade, ventricular end-diastolic pressure remains elevated and the atrial pressure waveforms change to those constriction, i.e. Y descent becomes prominent[20].

Management:

The treatment of cardiac tamponade centers around removal of fluid fromthe pericardial cavity and prevention of fluid reaccumulation. In the absence of tamponade related to continued bleeding into the pericardium from trauma or perforation of cardiac or aortic structures, pericardiocentesis alone may be sufficient as the initial treatment. Pericardiocentesis also afforts an opportunity for assessment of the fluid to aid in the diagnosis of pericardial pathology. Pericardiocentesis is generally performed under local anaesthesia, and premedication with atropine may reduce vasovagal symptoms that occur in some patients. The safety and success of pericardiocentesis are improved when simultaneous echocardiographic and electrocardiographic guidance are used[45].

Hemodynamic improvement generally occurs with withdrawal of the first 50 to 100 ml of fluid as the steep portion of the pericardial pressure-volume relationship decreases. Once pericardial pressure falls below the right atrial pressure, continued withdrawal of fluid leads to no further hemodynamic improvement. Following pericardiocentesis, continuous drainage may be facilitated by an indwelling catheter with multiple side holes connected to a closed drainage system.

Vasoactive agents and volume loading are generally ineffective in producing significant improvement in cardiac output and should be used, if at all, only in preparation for pericardiocentesis[7,22].

Limited subxiphoid pericardiotomy done under local anesthesia is a valuable alternative to percutaneous pericardiocentesis and offers the additional advantage of a pericardial biopsy[1]. However, a more thorough and radical surgical pericardiotomy is more appropriate for patients with purulent pericarditis because of enhanced drainage and the reduction of the otherwise high risk of constrictive pericarditis.

When bleeding into the pericardium from trauma or spontaneous rupture of cardiac chamber or aorta is the supected culprit, immediate thoracic surgery is essential for relief of tamponade as well as for repair of the source of bleeding. In these patients, percutaneous pericardiocentesis alone is rarely successful but may be attempted in preparation for immediate surgery.

Prevention of Recurrent Effussions

Recurrent pericardial effusion and tamponade is generally managed with surgical pericardiectomy. However, intrapericardial steroids have been used, particularly in dialysis patients, to reduce the need for pericardiectomy. Intra-pericardial tetracyline and chemotherapeutic agents are used for recurrent malignant pericarditis.

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Leading Articles

CORONARY ARTERY BYPASS GRAFTING VS. PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

*PRADEEP JAIN & UPENDRA KAUL .

Coronary revascularization is a well known method of treating a large number of patients with coronary artery disease. The two well accepted methods of achieving revascularization are 1) the surgical procedure of Coronary Artery Bypass Craft Surgery (CABG) - a technique which started in late 1960"s and 2) non-surgical technique of Percutaneous Transluminal Coronary Angioplasty (PTCA) which began in 1977-78[2]. Although the technique of surgical revascularization is 10 years older than PTCA, a large number of patients are being subjected to non surgical revascularization. In the USA, it is estimated that PTCA is now carried out more frequently than CABG, accounting for approximately 58% of all coronary revascularization procedures[3]. In India, the figures are however different with the figures of 1994-95 being 5,500 PTCA's Vs. 1100 CABG's (ration 1:2)[4].

PTCA is a very well accepted method of revascularization because it is non surgical, there is no requirement of thoracotomy, blood transfusion and a very short hospital stay. The technique to begin with was only applied to selected cases of single vessel disease (isolated, discrete, concentric lesion 'Gruentzig' lesion) and has been shown to be more effective than medical treatment with regard to clinical symptoms in patients with single vessel disease[5]. In recent years, the application of angioplasty has been extended to certain patients with multivessel disease - an approach stimulated by advances in balloon catheter techniques and increased expertise in performing the procedure[6-10]. The current success rates for properly selected cases is in the range of 90-95. Several non balloon techniques like directional atherectomy, rotional atherectomy, laser angioplasty and intracoronary stenting have expanded the indications for angioplasty. Patients can now be taken up for PTCA with good acute results and minimal short term complications.

Despite the technical advances in the methods of PTCA, one problem which has not found a satisfactory solution is the problem of restenosis which develops in 20-20% of patients within 3-6 months of the procedure[11-12]. The newer techniques of directional atherectomy, rotational atherectomy and laser angioplasty have not been able to reduce the incidence of restensos[13-15].owever, in the last few years, the experience with intracoronary stenting in de novo lesions has been revealing in that the restenosis rates have beejn shown to be significantly reduced[16-18]. Deployment of stent after PTCA seems to be a very promising method of achieving an excellent short term and long term result.

Till recently long term results of coronary angioplasty have been vailable only from registry studies[19-21] whereas in contrast coronary artery bypass surgery is an established treatment whiich has beejn the subject of several randomised clinical trials[22-24]. CABG has been demonstrated to produce an imprroved survival in certain subset of patients: 1) left main disease; 2) Three vessel disease

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* Department of Cardiology
C.T. Centre
All India Institute of Medical Sciences,
New Delhi-110029

with left ventricular dysfunction; 3) Three vessel disease with proximal left anterior descending artery involvement. Like PTCA, the results of CABG are also steadily improving with the routine use of arterial conduits,antiplatelet therapy and better intra operative protection of heart[25]. No doubt, surgery is a more in invasive procedure with a prolonged hospitalization and recovery time but the long term results are very gratifying. Although a large percentage of venous grafts get diseased over a period of 10 years, arterial grafts (the left and right internal mammary artery, gastroepiploic artery and radial artery have an excellent long term patency record[25]

THE CHOICE OF PTCA Vs. CABG

How should we treat patients who are suitable for either method of myocardial revascularization: Are the immediate risks, long term benefits, and total costs of the two treatments similar, or does coronary angioplasty offer short term convenience at the expense of long term efficacy? The only reliable method for comparing the two coronary revascularization procedures is a randomized clinical trial. Five such trials have been initiated. The results of three of these - Multicentre Randomised Intervention Treatment of Angina (RITA),¦The German Angioplasty Bypass Investigation (GABI) and the Emory Angioplasty Surgery Trial (EAST) have been published[26-28] and are discussed (Table-1). The results of the European Coronary Artery By-pass Revascularization Investigation (BARI) trial are awaited.

Table No :-1. Results of the Rita, Gabi & East Trails.

	RITA		GABI		EAST	
	PTCA	CABG	PTCA	CABG	PTCA	CABG
NUMBER OF PATIENTS	510	501	132	177	198	194
EARLY RESULTS						
DEATH	0.8%	1.1%	1.1%	2.5%	1.0%	1.0%
MYOCARDIAL INFARCTION	3.5%	2.3%	2.3%	8.1%	3.0%	10.3%
LATE RESULTS						
FOLLOWUP PERIOD(YEARS)	2.5	2.5	1	1	3	3
DEATH	3.1%	3.6%	0.8%	0.8%	7.1%	6.2%
MUOCARDIAL INFARCTION	6.6%	5.2%	2.0%	-	14.6%	19.6%
PTCA	18.2%	3.1%	23%	5%	41%	13%
CABG	18.8%	0.75	18%	1%	22%	1%
ANGINA FREE	69%	78%	71%	74%	80%	88%

With the exception of RITA, the trials exclude patients with signle vessel disease, which sunport the widely held but unproven view that coronary angioplasty is the treatment of choice for these patients. In a recent study[29] where the outcome of CABG was compared with in patients with isolated proximal left anterior descending artery stenosis it was ocncluded that both CABG and PTCA improve the clinical status of symptomatic patients with single vessel coronary artery disease and if the risk of restenosis and reintervention associated with PTCA is acceptable, it remains a suitable and simpler initial alternative to CABG.

The RITA trial[26] reported a 2.5 years follow up on 1011 patients randomised to either CABG or PTCA. The predefined combined primary even of death or definite myocardial infarction showed no evidence of a treatment difference (43 CABG, 50 PTCA). 4% of PTCA patients required emergency CABG beforhe discharge and a further 15% had CABG during follow up. Within 2 years of randomisation 38% and 11% of the PTCA and CABG groups, respectively, required revascularization procedures (s) (p<.001). The prevalence of angina during follow up was higher in the PTCA group (32%) Vs. 11% at 6 months) but this difference became less marked after 2 years (31% Vs. 22%).

The GABI trial[27] reported the outcomes at one year in 359 patients randomized to either CABG or PTCA. Hospitalization was longer after CABG (19 days Vs. 5 days for PTCA). Q wave myocardial infarction was more frequent with CABG (8.1% Vs. 2.3% with PTCA) whereas in hospital mortality did not differ significantly between the two groups (2.5% in CABG group and 1.1% in PTCA group). During the first year of follow up further interventions were necessary in 44% of the patients in the PTCA group (repeat PTCA in 23%. CABG in 18% and both in 3%) but in only 6% of the patients in the CABG group (repeat CABG in 1% and PTCA in 5%) ($p<.001$). 74% of the patients in the CABG group and 71% of those in the PTCA group were free of angina one year after the treatment.

The EAST trial[28] reported a 3 year follow up on 392 patients randomised to either CABG or PTCA. The composite primary end point of death, Q wave myocardial infarction and a large ischemic defect identified on thallium scanning at three years was similar in the CABG and PTCA group (27.3% Vs. 28.8%). At three years, the proportions of patients in the CABG group who required repeat CABG (22 and 41% respectively ; $p<.001$) Angina was more frequent in the PTCA group (20%) than in the CABG group (12%).

All the above trials have given the same answers viz. no significant difference in the risk of death or myocardial infarction, recovery after CABG which is the more invasive procedure takes longer with longer hospitalization than after PTCA and repeat procedures (PTCA or CABG) are much more common in the angioplasty group. Therefore, in multivessel disease, the patient's wishes should be strongly considered in selecting a procedure for revascularization. For patients who wish to avoid surgery, angioplasty is a reasonable alternative with no higher risk of morbidity or mortality than that associated with surgery. Patients must be prepared however for the possibility that additional revascularization procedures may be required after angioplasty in a significant number of cases.Conversely, patients who prefer the more definitive therapy can undergo surgery without fear of increased mortality of morbidity. However these trials comparing the results of CABG with PTCA were in the late 80's and with the advent of newer techniques like intracoronary stenting with lower restenosis rates, the comparative outcomes would be different in 1995. At the present time the focus of treatment should be on effective and long term revascularization. CABG and PTCA are two different methods with a common goal. The choice of therapy has to be individualized. The limitations and short comings of both methods should be realized.

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Leading Articles JK Practitioner 1996; 3:1 23-27

NIMODIPINE FOR TREATMENT OF ISCHAEMIC STROKE

Shafiqa A. Tanki*, Yasir A. Qazi, Zubeeda Jeelani***

SUMMARY:

Stroke research has achieved many important advances in the area of pharmacological intervention. Nimodipine has proven to be an effective treatment for ischaemic stroke. Its dual action with cerebrovascular and neuronal calcium ion channels has proven effective for treatment of ischaemic complications in patients with aneurysmal subarchnoid haemorrhage (SAH) and also appears effective for patients with non-haemorrhagic cerebral infarction.

Key words: Nimodipine Ischaemic Stroke.

INTRODUCTION

Acute ischaemic cerebrovascular disorder (ICVD) is a common devastating disorder without beneficial therapy. Treatment to prevent such damage in based on better understanding of the changes in patho-physiology during and after ischaemia. Oxygen, glucose and high energy phosphate levels are equilibrium across the cell membrane demands enerby. In energy failure, tonic homoeostasis can no longer be maintained. Na^+ moves into, K^+ moves out of and Ca^{++} moves into the cell. The sodium influx causes intracellular oedema, and the sharp increase in intracellular oedema, and the sharp increase in intracellular calcium triggers a series of destructive events influencing the neuronal damage. Ischaemia induces the release of excitatory amino-acid neurotransmitters such as glutamate, asparate and glycine which promote calcium entry into neurons via receptor-mediated calcium channels such as the kainate, the alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) and the N-methyl-D-asparate (NMDA) channels[1,2]. Increased free intracellular calcium in the neuron due to the disruption of calcium homeostatic mechanism leads to depletion of high energy phosphates, ever-ectivation of phospholipases and proteases, degradation of the neuronal cell membrane, production of cytotoxic free radicals, leukotrienes and prostaglandins. Increased intracellular calcium also causes uncoupling of oxidative phosphorylation and inhibits cellular respiration, leading to irreversible cell dysfunction and subsequent neuronal death[3,4]

The MDA channel has at least 6 sited which may be succeptible to pharmacological blockade[4]. The glutamate recognition site has been blocked experimentally by the compound CGS-19755 the glycine site by HA-966, the "Upper Competitive Site" by MK-801, NS-1102, and d-Methorhan, the polyamine site by Ifenprofil and another competitive site of Mg^{++}. As a sixth site - a site of phosphorylation - no antagonist has yet been identified. Enzymatic inhibition of nitric oxide synthetase by N-nitro-L-arginine also appear to protect against glutamate neurotoxicity[5]. Several AMPA antagonists were observed to reduce infaret volumes. Side-effects such as hypotension, sedation and nephrotoxicity were observed with some of these agents. Efficacy of these agents in humans awaits clinical trials.

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*Department of Clinical Pharmacology
SK Institute of Medical Sciences, Srinagar
**Department of Radiotherapy
Government Medical College, Srinagar

Address for Correspondence and reprints:
Dr. Shafiqa Aslam
Associate Professor
Clinical Pharmacoloty
SK Institute of Medical Sciences, Srinagar-190001.
Post Bag NO: 27

Voltage, dep[endent calcium channel antagonists are the only agents which have reached the clinical trials stage. Verapamil, Diltiazem and Nifedipine have poor brain penetration and have never been considered viable candidates for cerebral protection. Nicardipine was evaluated in a small phase I trial. Thirty five patients were randomized to received 3-7mg/kg/hour IV infusion versus placebo[6]. All patients showed improvement with a slight difference favouring nicardipine. No further studies were performed.

The greatest experience to date has been with nimodopine, a voltage sensitive second generation calcium antagonist which has proven to be an effective treatment for ischaemic stroke and significantly reduces mortality[7,8]. A dihydropyridine, nimodopine is nifedipine like in structure, a highly lipophilic and crosses the blood brain barrier in high concentration. In central nervous system, high affinity binding sites (receptors) to nimodipine have been identified in vitro[11], having different densities in different parts of CNS, preferentially in the hippocampus and cortex. Thus binding of nimodipine to these receptor sites regulates the influx of calcium through its slow calcium channels and these properties explain efficacy in the prevention and treatment of ischaemic neurologic deficit.

Pharmacodynamic and Pharmakokinetic Actions:

Nimodipine has an antivasoconstriction effect that is exerted in particular on the cerebrovascular system. The substance inhibits the transmembrane entry of calcium ions necessary for vascular muscle contraction. Experimentally, the cerebral vessels have proved significantly more sensitive to nimodipine, than peripheral vessels.

Nimodipine through an inhibition of accumulation of intracellular calcium has a neuroprotective or cytoprotective (tolerance to ischemia) effect against ischaemia[10,14]. The drug protects the badly damage nerve cells from being killed off and brings back the flow that they need to survive. Nimodipine also exerts neuropharmacological and psychopharmacological effect. In patients with acute cerebral blood flow disturbances, nimodipine has been found to produce a stronger vasodilation and to increase blood flow to a greater extent in damaged and hypoperfused regions of the brain than in normal regions[13]. Following subarchnoid haemorrhagea, the excess blod acts like a toxin and causes spasm and constriction of the vessels leading to neurological damage. Nimodipine helps to prevent the spasm and the mortality rate is significantly reduced. Vasoconstriction induced by various other vasoactive substances such as serotonin prostaglandin, can also be eliminated or prevented by nimodipine.

After oral administration, nimodipine undergoes rapid release and is absorbed to an extent of more than 90%. Nimodipine has distinct pharmacokinetic characteristics which can be attributed to high brain blood ration of the drug and longer half life in the brain compared with the blood and this property might contribute to its neuroprotective effect. After biotransformation, the elimination takes place mainly via the bile. No loss of efficacy is observed in the course of prolonged use. Tolerability to the substance is good following both prophylactic and therapeutic administration.

Clinical Efficacy:

A major goal in the treatment of acute ischaemic stroke is to minimize the irreversible brain damage.

One strategy of preserving neuronal function is to increase cerebral blood flow[13] and another is to block the damaging and possibly irreversible disruption of cellular hemostatis that results from failure of energy dependent membrane pumps[14].

The belief that cerebral tissue may be salvaged after stroke is based on the concept of an ischaemic penumbra[24]. After occlusion of a cerebral vessel, some flow is maintained through collateral vessels and there are two critical thresholds for this flow[26] an upper level below which irreversible cell damage occurs. At flow rates between the two thresholds, neurons do not function but they are viable, and these cells constitute the ischaemic penumbra. The survival time or the time window (i.e. time between the onset of stroke and initiation of therapy) of ischaemic penumbra is limited, but cytoprotection or increasing blood flow within this period may limit the volume of infarction.

A number of therapies have been recommended, but as yet, no specific therapeutic regimen has proved to be of maximum benefit. The important role of calcium influx in cell death during cerebral ischemia has been known since late 1970's. This has been the theoretical basis for the clinical trials with nimodipine in the treatment of acute ischaemic stroke (Table I)[15]. The results o the earliest trials seemed to fulfill expectation[16-18], but later trials have for the most part provided negative results[8,19-22].

The largest trial conducted by the American Nimodipine Study Group[8] enrolled 1064 patients of 21 days in dose of 60mg, 120mg or 240mg per day compared with placebo. No significant differences in mortality or neurological function were seen in patients or neurological function were seen in patients treated within 48 hours of the stroke. The patients using 120mg/day showed decreased mortality when treatment was administered within 24 hours of stroke onset, but no significant difference in long term functional recovery. Sub-group analysis of those patients treated with 120mg/day within 18 hours of stroke onset showed significantly better outcome scores and a 30% reduction in frequency of deterioration. Benefit also correlated with negative initial brain C.T.

A meta-analysis of nine trials[23] totaling 3714 patients, indicated a benefit for those receiving early therapy. The 616 patients treated within 12 hours showed a pooled odds ratio of 0.62 favouring nimodipine (95% CL, 0.44-0.87)[11]. No effect on outcome was seen for age, sex, hypertension, diabetes or cardiac

Table-I **Earlier Placebo-controlled, double-blind studies on Nimodipine in acute ischaemic stroke**

Study	Year	n	Treatment started	Daily dose (mg)	Treatment period	Follow up	Case fatality	Functional outcome
Gelmers et al[16]	1988	186	<24 h	120	28 d	6 mo	N >> P	N >> P
Faci et al[17]	1989	41	<12 h	120	28 d	28 d	-	N P
Wimalaratna and[18] Capildeo	1989	215	<24 h	120/ 240	16 Wk	6 mo	120 mg>	240 mg> P
Bugousslavsky et al[19]	1990	52	<48	120	14 d	4 mo	-	N = P
Martinez-Vila et al[20]	1990	164	<48 h	120	28 d	28 d	N = P	N = P
Trust Study Group[21]	1990	1215	<48 h	120	21 d	6 mo	N = P	N < P
Horing et al[22]	1991	482	<48	120	21 d	21 d	N = P	N = P
American Nimodipine[8] Study Group	1992	1064	<48	60/ 120/240	21 d	6 mo	N = P	N = P
Markku et al[15]	1994	350	<48 h	120	21 d	12 mo	N < P	N = P

N >> P indicates nimodipine statistically better than placebo; N>P, nimodipine nonstatistically better than placebo; N=P, nimodipine and placebo equivalent; and N<P, placebo nonstatistically better than nimodipine.

disease. There was no drug effect for those treated 13-24 hours, and a slightly worse outcome for those treated for 24 hours. The results of the most of these clinical trials of nimodipine have given promising treatment results[16-18]. For example, moderately severe or severe strokes might benefit from nimodipine treatment and men may respond better than women, a daily dose of 120mg may be better than 240mg treatment started within 12-18 hours after onset might be advantageous and a normal C.T scan obtained within 18 hours after onset seems to have positive effects on the results of nimodipine treatment. Several other clinical trials of nimodipine[27] have demonstrated benefit when therapy is started within 6-8 hours of an acute ischaemic event, However, the guidelines produced by the National stroke Association (NSA) in the United States has prepared the consensus statement to bring attention to the importance of the first minutes and hours after onset of stroke. By and large, it has been broadly agreed to obtain maximum benefits, nimodipine should be started within 6 to 8 hours of an acute ischaemic event (i.e. when the therapeutic window is open[21-26]).

Conclusion:

Treatment of stroke patients with nimodipine affectively places damaged cells in a state of 'Suspended Animation' providing time for recovery[11]. While earlier trials with the drug and not appeared very successful, this was probably related to the time elapsed after the stroke before treatment was administered. There is real evidence to support the use of calcium channel blocker nimodipine within 'Open Time Window' period of 6-8 hours for therapy in patients with ICVD apart from routine principles of best medical care to avoid cardiovascular complications. This required that the patient arrives at the hospital early and that treatment is started without delay. With patient, paramedic and physician education, this may be achieved[17].

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"LAPAROSCOPIC CHOLECYSTECTOMY - AN EARLY EXPERIENCE"

***Nazir Ahmad Wani, **Tajamul Fazili, **. Omar Javed Shah.**

Despite the development of many non-operative therapeutic options for symptomatic gallstones, cholecystectomy, first performed by Carl Langenbuch in 1992, still remains the preferred form of treatment. Extra corporeal Shockwave Lithotripsy (ESWL) and/or oral dissolution therapy are recent developments but are applicable to only 15-20% of patients. Furthermore, even after successful stone fragmentation and clearance, recurrence occurs in more than 50% patients [1,2].

Until recently, Cholecystectomy required a Laparotomy. Disadvantages of Laparotomy include post-operative pain, transient paralytic Ileus, pulmonary complications and wound problems in addition to lengthy hospitalization and prolonged recovery period. Laparoscopic Cholecystectomy (LC) was first performed by Phillipe Mouret in Lydan, France in 1987. Within a year this procedure became widely accepted in the World. Advantages of LC which became apparent even in small series of case have been confirmed by larger studies and include diminished post-operative discomfort, dramatically reduced hospital stay and shortened recovery period. There is now evidence that percent reduction in pulmonary performance is smaller after laparoscopic than traditional or mini-incision cholecystectomy [3] and return of intestinal motility is faster [4]. With escalation of interest in LC, knowledge about the instruments and procedure is becoming essential. Besides, selection of patients, results and complications are other important considerations.

Equipments and Instruments:

The various equipments and instruments used in LC include:

1. High resolution video camera and monitor to enable the whole team to follow the procedure simultaneously.
2. Light source, which is a 50 Watt metal halide lamp providing bright white light.
3. High flow Insufflator which can deliver upto 10 liters of carbon dioxide per minute.
4. Suction - irrigation device which allows intermittent irrigation and suction of operative field.
5. Telescope with zero degree viewing angle and 10 mm diameter.
6. Verses pneumoperitoneium needle.
7. Four Cannulae to act as ports for introduction of telescope and other instruments.
8. Grasping and dissecting forceps.
9. Scissors.
10. Clip applicator for occluding cystic artery and duct by titanium clips.
11. Elector-surgical Unit is a unipolar elector-cautery system to ensure hemostasis throughout the procedure.

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* Professor & Head.
** Lecturer.
DEPARTMENT OF GENERAL SURGERY,
SK INSTITUTE OF MEDICAL SCIENCES,
SRINAGAR, KASHMIR.

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Sterilization and Maintenance:

All the hand instruments, including telescope and camera are disinfected by soaking in activated Gluteraldehide solution for 25-30 minutes. Instruments are thoroughly cleaned after the procedure and then dried.

Indications and patient selection:

Indications for LC are the same as for traditional surgery. The contra-indications include:

A) Absolute

01. Unfit for general anaesthesia.
02. Bleeding disorders.
03. Pregnancy.
04. Jaundice at the time of surgery.
05. Common duct stone with failed Endoscopic sphincterotomy.
06. Suspected Carcinoma of the gall bladder.
07. Generalized peritonitis.

B) Relative

01. Acute cholecystitis.
02. Previous multiple abdominal surgery.
03. Portal hypertension.
04. Cholecystoenteric fistula.
05. Morbid obesity.

with increasing experience, the contra-indications are decreasing.

Pre-operative evaluation:

A detailed history should confirm that the symptoms are due to gall stones. An ultrasound examination must confirm the presence of gall stones and assess the diameter of the CBD and other pathology besides evaluating the pancreas.

The patient should be fit for general anaesthesia. Elderly patients with higher risk due to Cardiac and Pulmonary disease are better served by traditional surgery. Patients who are offered LC should be specifically informed that if any technical difficulty is encountered, the procedure may be converted to open cholecystectomy for hid/her safety.

Experience at SK Institute of Medical Sciences, Srinagar

From April, 1995 to November, 1995, 39 patients underwent LC. All of them were females age ranged from 18 years to 59 years, with a mean of 38.5 years. All patients had symptomatic gall stone disease (Table-I) eight (19.3%) of our patients were over weight and six (14.6%) had scars of previous abdominal procedures. LC could be performed in 31 cases while conversion to open procedure was required in 8 (20.5%). The reasons for conversion are shown in Table-II. Operating time ranged from 45 minutes to 120 minutes with a mean of 83 minutes. Complications occurred in 2 patients and included biliary leak which was detected by bile drainage through the Redevac suction drain, (which we use routinely) and settled on the 3rd post-operative day. No patient required exploration and there was no mortality. Mean hospital stay was 2.5 days. 14.3% were discharged on the 2nd post-operative day and 86.6% on the post-operative day.

Discussion

After open cholecystectomy (OC), an average hospital stay of 3-4 days and a recovery period of 4-6 weeks is expected, before resumption of normal activity. Laparotomy is associated with significant post-operative pain and discomfort and may be attended by chest complications would infection, incisional hernia and scar hypertrophy.

LC is an attractive alternative to OC for definitive treatment of symptomatic gall stones.

Post-operative pain and need for analgesics is greatly reduced; as are pulmonary complications. Average post-operative stay after LC is 2-3 days [5,6,7]. Recovery is faster and most patients return to normal activity in a weeks time [7,8]. Infection and other wound problems are virtually eliminated. The ultimate cosmetic results are very good, particularly since the umbilical incision can be so placed that it will subsequently disappear inside the umblical fold.

The most serious concern about LC has been reports of a slightly higher incidence of CBD injury. In OC, duct injury occurs in approximately one in 400 cases [9]. In our series of 39 cases, we had no ductile injury, though reports suggest that duct injury occurs in 0.5 to 2% of LC's [5,8]. Our conversion rate at present (20.5%), which is higher as compared to the reported figure of 5% could be attributed to our early experience in this field.

During LC certain complications can occur which are unique to this procedure. These include bowel or gastric perforation by verses needle or trocar, thermal injuries to the colon or duodenum, haemorrhage or bile leak from improperly applied clips and spill of bile or stones into the pertioneal cavity. These complications are infrequent and the risk is out weighed by the low rate of other complications [5,7].

Future

It seems clear that LC is a useful procedure. It has significant advantages over OC and it represents a permanent advance in the management of cholelithiasis. It is a new procedure and results are likely to improve further with accumulation of experience and improvements in the design of optic and other instruments. Though termed as minimal invasive surgery, it can cause complications similar to or even worse than OC, particularly in in-experienced hands.

TABLE-I

S.No:	Indications	No. of Patients	Percentage
01.	Calculous chronic cholecystitis	33	84.6
02.	Mucocele of the Gall bladder	04	10.2
03.	Asymptomatic gall stones.	02	5.2

TABLE-II

S.No:	Cause	No. of Patients	Percentage
01.	Difficult dissection	5	62.5
a)	Deuse adhesions	3	
b)	Obscure anatomy	3	
02.	Bleeding	2	25
03.	Grossly Inflamed organ.	1	12.5

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Intra-Ocular Lens Implantation Surgery

*Sheikh Sajad Ahmad

The changes that have occurred in cataract surgery over the past 25 years have been voluminous. If master surgeons of yesterday could return and see how cataract surgery is performed today their amazement would be indescribable. In 1745, Jacques Daniel performed first extracapsular cataract extraction (Arthur Lim). His technique was used upto 20th century. The first persons who probably mentioned the possibility of lens implantation was a peripathetic Opthalmologist of the 18th century named Tadini. In 1766, Casanova met him in Warsow. Tadini showed him a box with small spheres that were well polished. They soon realized, however, that the glass lens could not substitute for the natural lens because during his experiment, the glass fell on to the botton of the eye (Alpar and Fechner 1988).

In 1949, the first practical implant was conveived, it was inserted in the posterior chamber, occupying nature both anatomically and optically as closely as possible.

The first intraocular lens was a posterior chamber intraocular lens which was inserted on November 29th, 1949 by Harold Ridley. It weighed 106 mgm in air. It seens to have commenced with observation that particles of poly-methylmethacrylate from the canopies of British Spitfire planes during the World War II occuasionally penetrated the eyes of pilots and incited little or no inflammatory re-action. Some years later in the autumn of 1949, a medical student while observing Ridley remove a cataract at St. Thomas Hospital in London is said to have remarked that he had forgotten to replace the diseased lens with a new one. Ridley already primed by the observation of the inert plastic within the pilots eyes proceded with his forst implantation. Becuase of an error in optical calculation the patient became highly myopic. This early design proved faulty, with dislocation both anteriorly and posteriorly into the vitreous. This being the most serious complication, the procedure was abandoned (Alpar and Fechner 1988).

The multiple difficulties with early posterior chamber lenses lead to abandonment of the posterior chamber as the site of pseudophakos. First inserted by Baron of France in 1952, enterior chamber fixation next captured the interest of implant pioneers. Inspite of formidable obstacles and professional criticism choycs of England persisted with the anterior chamber imnplants. Iris supported lenses also did not achieve much recognition and finally modern era of posterior chamber lens implantation started again. Pioneers in this field include J.C. Pearce, E.I. Arnott, W.S. Harris, J.S. Cittle, Q.W. Simcoe, S.P. Shearing and Sinskey. In 1975, John Paerce of England became the first surgeon to return to the posterior chamber intraocular implants (Steven Shearing 1982).

In March, 1977, Shearing introduced the first J-loop posterior chamber lens. Almost immediately this lens design captured the imagination of implant surgeons as it fulfilled Ridley's prophetic words nearly 30 years age. The flexible loops could be compressed to render the lens small during insertion and upon placement in posterior chamber of loop, allowed expansion to provide stability. The

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* Registrar Deptt. Opthalmology
Medical College Srinagar, Kashmir
Pin - 190 010

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original J-loop style has received several modification.

The loops may possess an anterior 10 degree slant to permit further displacement of optical disc into posterior chamber, thereby lessening the incidence of pupillary entrapment behind the optics (Steven Shearing 1982).

The C-loop posterior chamber implant allegedly places less pressure outwards by the more curved loops than does the J-loop design.

Today the most common method of implant surgery is that of extracapsular cataract extraction with implantation of posterior chamber intraocular lens, as evidenced by the fact that in 1977 less than 1% of the total intraocular lens were posterior chamber whereas in 1984 they accounted for 20% of the total intraocular lenses.

Technological advances in extracapsular cataract extraction with the use of Healon, Chondroitin sulfate, and methyl cellulose have edded to the success of the procedure.

Complications of intraocular lenses (Abhay 1991):

1. **Corneal**
   - a. Stripping of Descemet's Membrane.
   - b. Intraocular lens corneal touch.
2. **Uveal complications**
   - a. Pigment disparsion
   - b. Iris Chafind.
   - c. Micro hyphaema syndrome/visual white out
   - d. Distorted pupil
   - e. Pupillary capture or iris entrapment
   - f. Moisis
   - g. Mydriesis
   - h. Break down of Blood-*Aqueous Barrier*
   - i. Fibrinoid rections
   - j. Cocoon membrances
   - k. Chronic pupillary membrances
3. **Post implantation Glaucoma:**
   Causes include:
   - a. Pupillery block
   - b. Angle blockage and peripheral anterior synethiae
   - c. Pigmentary glaucoma
   - d. Steroid induced glaucoma
   - e. Transient ocular hypertension
4. **Post implantation inflammation and clinical Toxic syndrome:**
   - a. Intraocular lens related inflammation
   - b. Inflammation not directly related to intraocular lens.

   a. Intraocular lens related inflammations:
   Its causes include:
   Mechanical factors: related to lens types, designs, fixation and manufacturing defects include:
   Uveitis, Glaucoma and Hyphema (UGH) and UGH+Syndromes (Ellingson'a Syndrome).
   Irritiating compounds on intraocular lens:
   Residual polishing compounds on intraocular lens
   Intraocular sterlization problems:

   b. **Inflammations not related to intraocular lens:**
   Infectious Endophthalmitis
   Non-infections Endophthalmitis
   Phaco-toxic and Anaphylactic Endophthalitis
   Sympathetic Ophthalmia
5. **Pseudophakic Endophthalmitis**
6. **Malpositions of posterior chamber intraocular lenses:**
   It includes:
   a. Loop malpositions
   b. Decentration of the optic
   c. Sunset syndrome
   d. Sunrise syndrome
   e. Windscreen wiper syndrome
   f. Horizontal displacement
   g. Lost lens syndrome

7. **Opacification of posterior capsule:**
Its aetiology includes:
a. Thickened left over fibres
b. Plaque
c. True posterior capsular
d. Fibrinous membrane
e. Pigment despsition
f. Wrinkled capsule
g. Anterior capsular opacification
(Abhay 1991)

8. **Retinal complications:**
These include:
a. Cystoic macular oedema
b. Rational detachment
Both the complications are secondary ot barrier Deprivation sydnrome (Aipar and Fechner 1988) common with anterior chamber implants.
c. Posterior damaging effects of light on Retina - Light Maculopathy (Erythropsia) (Alpar & Fechner 1988)

9. **Post-operative Astigmatism**

10. **Wrong power intraoculaer lens**

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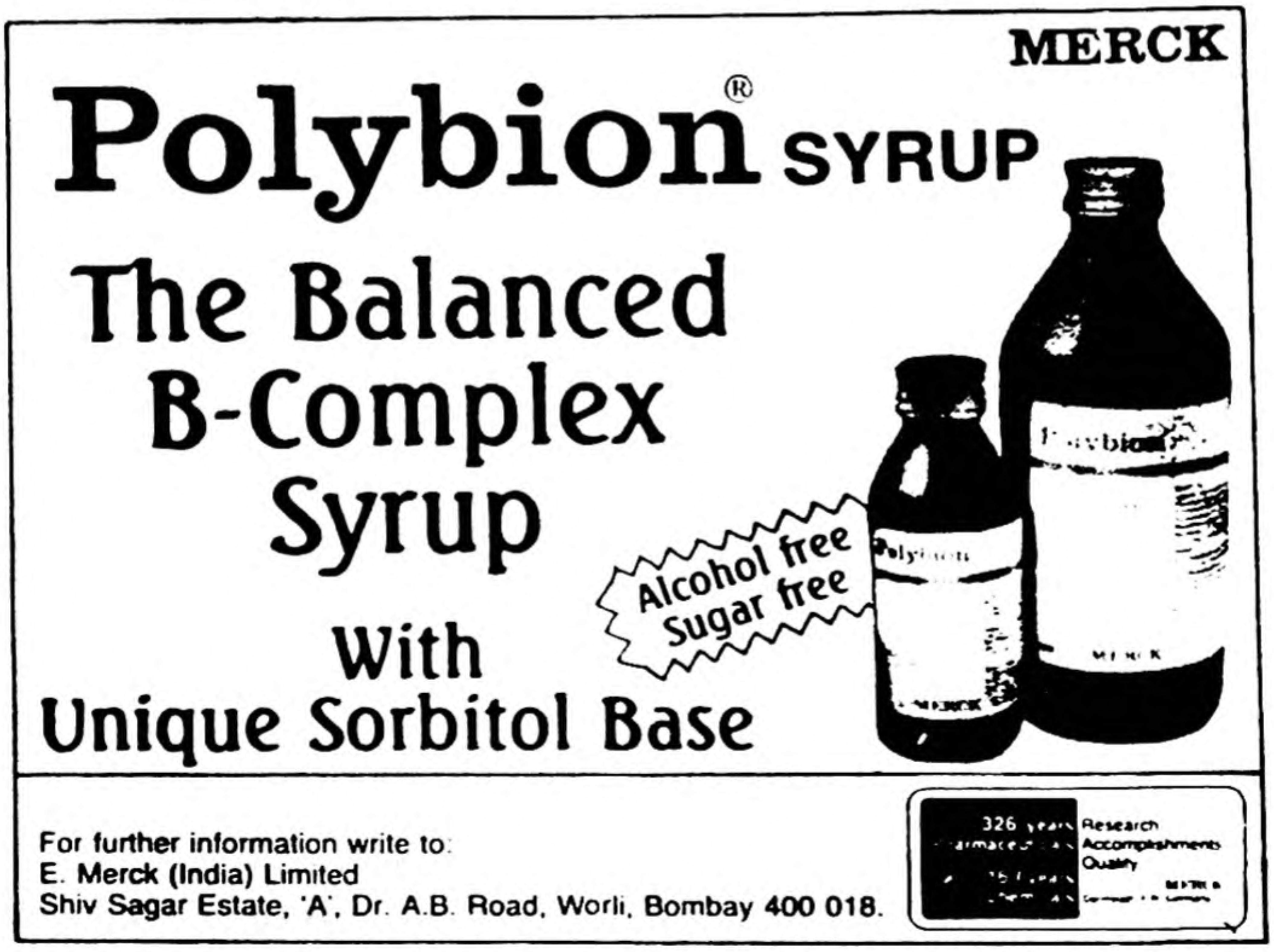
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JK Practitioner 1996; 3:1 34-36

HEARING IMPAIRMENT IN SCHOOL CHILDREN OF KASHMIR "AN EPIDEMIOLOGICAL PROFILE"

ZAHOOR AHMAD*; ABDUL AHAD**

INTRODUCTION

Hearing loss is an imprtant contemporary problem in child health. Screening of children for hearing problems has been carried out for over 50 years in Schools of various Countries. There is an urgent need for school health maintenance programmes for developing countries like India which are striving to make rapid progress with special emphasis on health services in order to achieve the WHC's goal of health for all by the year 2000.

The evaluation of auditory problems is accomplished through the use of otoscopic examination, auditory threshold testing and tympanometry; while as the most common cause of ear disease in children is medically treatable middle ear disease[9].

This report reviews 386 school children with documented hearing loss evaluated at SMHS hospital Srinagar, Kashmir after preliminary screening of 11000 school children (males 5500 : Females 5500) from 4 to 14 years of age among 50 schools within various localities of Srinagar city. Factors examined include demographic variables, socio-economic data, presumptive etiology as well as severity. The prevalence of school related problems among the study population is also addressed.

MATERIAL AND METHODS

This study was conducted from June, 1992 to December, 1993 in 50 schools of Srinagar city. During this period 11000 school children (5500 males : 5500 females) were examined. These children were between 4 and 14 years of age. After obtaining relevant history regarding probelms relating to ear, nose and throat these children were subjected to general physical examination which was followed by otoscopic examination and tuning fork tests. Out of 11000 children 386 children were found to have hearing impairment and/or otological pathology sufficient to warrant further evaluation in ENT Department of SMHS Hospital Srinagar to categorise the hearing loss and to establish its exact etiology. A detailed clinical history was taken from parents of each child in addition to getting information about demographic and socio-economic data. This was followed by detailed ENT examination of these 386 children who where investigated as folloes:

Pure tone audiometry in case of all 386 children, impedance audiometry in case of 268 children who had intact tympanic membranes excepting 8 cases of acute suppurative otitis media, vestibular evaluations (cold caloric test and positional test) and BERA in case of children suffering from sensorineural hearing loss, X-ray nesopharynx in case of children suffering from otitis media with effusion and X-ray mastoids in cases who were suffering from chronic suppurative otitis media.

RESULTS

Out of 11000 children screened by otoscopy and tuning fork tests in schools 386 children who were found to have hearing impairment and/or otological pathology were studied further for establishing underlying etiology. 235 (61%) were males and 151 (39%) were females with a male-female ratio of 1.6 : 1.302 cases had bilateral hearing loss and

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* Registrar
** Professor and Head,
Department of Otorhinolaryngology (ENT),
Govt. Medical College, Srinagar.

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eighty four cases had unilateral hearing loss. About 60 percent cases were suffering from mild to moderate hearing loss.

Socio-economic status had a direct relationship with hearing loss, as it was found commonest among lower strata of society i.e., 60.7 percent. Environmental factors also were found to have direct bearance on ear disease as 228 (59 percent) children belonged to congested localities whileas only 158 (41 percent) children belonged to non-congested localities.

As regards the academic achievements, when the children with hearing loss (386) were compared with controls (10614) the mean percentage of marks was 31.8 percent among the former group and 51.4 percent among the later group, which is statistically significant.

Tympanometry was performed on 268 children. 118 children were not subjected to this test and they included 104 cases of chronic suppurative otitis media, 4 cases of traumatic perforation, 8 cases of acute suppurative otitis media and 2 cases of meatal atresia.

Ears showing type As, B and C curves were pathological and ears showing A and Ad curves were normal, the later resulting from flacidity of normal tympanic membrane.

Stapedius reflex was absent in 222 ears out of which 218 ears suffered conductive loss and 4 ears suffered profound sensorineural hearing loss. On cold caloric test only 3 children out of sensorineural hearing loss cases had hypoactive labyrinth.

BERA revealed waves of low amplitude and delayed latency in 2 cases of SNHL out of whom one had suffered severe perinatal asphyxia and other had congenital sensorineural hearing loss.

On radiological examination 4 cases showed cholesteatoma in X-ray of mastoids and 88 cases showed adenoid hypertrophy in X-ray of nasopharynx.

364 children were suffering from conductive hearing loss (Figure-I_. Secretory otitis media accounted for about half of the cases (47.9 percent) of conductive deafness. 19 children were suffering from sensorineural hearing loss (Figure-II). Among them in about 1/3rd of cases meningitis was the underlying case and in another 1/3rd of cases no cause could be found. 3 children were suffering from mixed hearing loss out of whom 2 children had chronic suppurative otitis media and one child had Down's syndrome.

DISCUSSION

In the present study 3.5 percent prevalence of hearing loss was found which goes in agreement with most of the workers (3.7 percent[3], and 3.5 percent[10]). Our prevalence rate is higher than that found by previous workders (.3 percent[3], 2 percent[7]) in British School Children which can to. due to low standard of living in our set up as compared to British population. The prevalence data available from longitudinal studies conducted in British School Children from 1911 to 1941[3] reveal a steep fall in incidence of deafness from 3.7 percent to 0.3 percent which can be attributed to improvement in their standard of living.

In our study we found overall tympanic membrane perforation rate of .97 percent (102 cases of CSOM out of 11000 children) which is consistent with previous findings (1 percent[12]), (<2 percent[8,11]). Conversely Australian Aborgiginal children have high incidence of tympanic membrane perforation i.e. 26 percent[5]. Our study advances the view poing that Kashmiri children are genetically less prone to otitis when compared with Australian Aboriginal children. It depicts that genetic factors intervent in determing susceptibility to ear disease.

A Close association is observed between hearing loss and school performance. Mean percentage of marks is 38.8 percent among hearing impaired and 51.4 percent among conrols. Out of the children suffering from sensorineural hearing loss 63 percent exhibited grade repetitions, a finding consistent with that of previous workers[2] who found 59 percent cases having wither academic or behavioural problems at shcool among their study population having sensorineural hearing loss. It shows that hearing loss, especially sensorineural type causes educational handicap to the child. These children need special educational help.

By classifying the hearing losses we found 94.3 percent cases having conductive deafness which is consistent with previous reports (92.8 percent[1]). Commonest cause of hearing loss in our study is otitis media with effusion (45 percent), which goes in agreement with the findings of previous workers (50.9 percent[1]).

Common cause of sensorineural hearing loss in our series is meningitis (31 percent) which concurs with the findings of most of the workders (24 percent[4], 40 percent[6]). In 31 percent cases of sensorineural hearing loss no definite cause could be detected. The presumptive aetiology in this idiopathic group as unrecognised cochlear pathogens, undiagnosed intrauterine infections or subclinical viral labyrinthitis in early infancy is yet to be established. A concerted effort at further research is warranted.

CONCLUSION

The present study reveals that in our set up otitis media with or without suppuration is the commonest cause of hearing impairment in school children i.e. 74 percent. This is due to a variety of reasons such as lack of adequate health services, low standard of living, lack of medical knowledge, poor hygienic conditions and climate but at the same time it gives hope that improving the health services by promoting community medicine and by employing well origanised pre-school and school hearing screening programmes the number of hearing impaired children might be reduced to a minimum. At this time there does not appear to be any prophylactic or therapeutic measure that would prevent those cases of sub-clinical viral labyrinthitis that occur in neonatal period and to achieve this goal a concerted effort at further research is warranted. Extensive efforts should be aimed at early identification of hearing losses because this determines the efficacy of management procedures. Otolaryngologists should lay emphasis on all sorts of requisite investigations to detect any auditory deficit at a very early age in case of high risk children.

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Original Article

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ACTIVATED CLOTTING TIME ESTIMATION IN HEPARIN REVERSAL DURING OPEN HEART SURGERY

*A. G. Ahanger, **Mehraj-ud-Din, *** Z. A Shah, **** A. M. Dar, *****Ishtyak A Mir.

Abstract:

The anticoagulant effect of heparin needs to be reversed with protamine sulphate in adequate dosage in open heart surgical procedures. Both under-or over-correction with protamin may lead to undesirable postoperative bleeding. We estimate ACT regularly during open heart surgery to determine the adequate dosage of protamine sulphate so that the ACT returns to base line levels. We have used slow controleed infusion of protamine in 300 patients of open heart surgery guided by repeated ACT estimations and 94% of our patients had an adequate heparin reversal by this method.

Key words:

CPB = Cardiopulmonary bypass.
ACT = Activated clotting time.
ECG = Electrocardiogram.
ABGA = Arterial blood gas analysis.
I/V = Intravenous.

Introduction

Postoperative bleeding is one of the most dreaded complication following open heart surgery. Protamine infusion for heparin reversal is often on the border line, both under and over dosage being possible. There are wide variations in the recommended dosages of protamine(1,2).

We have adopted regular estimation of ACT as our guide for such dosage to return the ACT to base line.

Material and Methods:

This study involved 300 patients undergoing CPB for open heart surgery from 1983-1994 (Table I). All these patients were monitored for ECG, arterial pressure, central venous pressure, esophageal and rectal temperatures, ABGA, hematocrit, serum electrolytes, urine output. A baseline ACT was obtained in all, using Haemachron equipment.

Heparin was administered I/V in the dose of 3 mg/kg followed by 1.5 mg/kg as and when indicated so as to maintain ACT at more than 480 sec. All these patients were cooled to 28-25° during cardiopulmonary bypass and rewarmed to 37°c core temperature at the end of surgery. With stable haemodynamics soon after weaning from CPB, protamine was given with a micro-infusion in 1:1 ratio for heparin reversal over a period of 10 minutes. Regular ACT estimations were made at 5,10 minutes of infusion. Protamin infusion was stopped soon after ACT returned to preheparin levels and appearance of clots in the surgical field. ACT estimations were repeated after 15 minutes and 30 minutes in case of unexpected ooze from surgical field and additional protamine given if needed. Postoperatively drainage was monitored and measured every hour for 24 hours.

Observations:

Of the 300 patients studied none had ACT to the baseline before protamine infusion. 80% of patients had return of ACT to baseline after infusion of about 75% of estimated dose

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* Assistant Professor and Head CVTS.
** Chairman Anaesthesia Deptt.
*** Lecturer CVTS.
**** Mch Resident CVTS.
***** Registrar CVTS.

Department of Cardiovascular and Thoracic Surgery (CVTS) and Department of Anaesthesiology and Critical Care Medicine.

SK Institute of Medical Sciences, Soura,
Srinagar, Kashmir-190011.

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of protamine (Table II) while 14% of patients needed the total estimated dose of protamine. 6% of the patients needed addtional 25% protamine. Clot formation was adequate in all the patients once the ACT returned to normal values.

Unexpected extra drainage was observed in 14% of patients over a period of 24 hours; our of whom 8% needed extra doses of protamine to control their bleeding while 6% needed re-exploration for surgical bleed, (Table III).

Discussion:

Anticoagulant effect of protamine in excess is well established[3], as is decrease in platelet number and function. This can aggrevate the thrombocytopenia following

Table-I

Spectrum of open heart operations involved

S.No:	Open heart procedure	No. of patients	%age
01.	ASD repair	168	56.00
02.	VSD repair	18	6.00
03.	TOF	8	2.66
04.	PAPVC	8	2.66
05.	TAPVC	1	0.33
06.	Valve replacement	94	31.33
07.	LAM	3	1.00

ASD = Atrial septal defect.
VSD = Ventricular septal defect.
TOF = Tetrology of Fallot.
PAPVC = Partial anomalous pulmonary-venous connection.
TAPVC = Total anomalous pulmonary venous connection.
LAM = Left atrial myxoma.

Table-II

Activated clotting time in seconds:

ACT in seconds Pts.	Prehaparin		during CPB		Postprotamine infusion 5 minutes		10 minutes	
	No.of Pts.	%	No.of	%	No.of Pts.	%	No.of Pts.	%
100-200	300	100	-	-	200	66.66	240	80
221-300	-	-	-	-	42	14	18	6
480-600*	-	-	300	100	-	-	-	-

*Heparinization before CPB to keep ACT between 480-600 seconds and after protamine infusion ACT valves desired to baseline i.e. 100-200 seconds.

Table-III

Postoperative drainage, first 24 hours:

Drainage (ml)	Addl. dose of Protamine needed in				Re-exploration for bleeding in	
	No.of Pts.	%	No.of Pts.	%	No.of Pts.	%
< 500 ml	240	80	-	-	-	-
500-100 ml	42	14	18	6	-	-
> 100 ml	18	6	18	6	18	6*

*6% of the patients needed re-exploration for surgical bleeding and did not have any benefit from extradose of protamine.

CPB[4]. Other undesirable effects of protamine on cardiovascular and pulmonary systems are well documented[5]. Adequate heparin reversal needs correct dosage of protamine as determined by ACT estimations since over dosage of protamine is equally problematic. Correct heparin-protamine ratio varies with different studies[1,2] various methods of protamine titration to determine the optimum heparin reversal have been described[1,3,6,7] while automated protamine titration method, though accurate is very expensive[8]. Slow and controlled infusion of calculated dose of protamine in our patients till the return of ACT to prehaprin levels was effective in most of our patients (94%) while it was not so in only 6% of patients, who underwent re-exploration for surgical bleed.

Well known heparin-rebound dose not contribute to any significant postoperative bleeding[9,10]. Serial estimations of ACT with slow, continous infusion of protamine can help in detection and neutralization of any remaining heparin in circulation. Cardiovascular stability especially in patients with poor L.V function can be maintained by slow infusion of protamine.

Conclusion:

We conclude that serial estimations of ACT are mandatory in optimal neutralization of heparin in the circulation after CPB, thereby avoiding under-or over-correction with untoward consequences. This method of heparin reversal is fairly adequate and promising.

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Address for correspondance & request for reprints:
Dr. A. G. Ahanger, Assistant Professor & Head,
Department of Cardiovascular & Thoracic Surgery,
SK Institute of Medical Sciences Soura,
Srinagar, Kashmir-190011
Post Bag NO: 27

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MIXED/COMBINED GERM CELL TUMOURS OF TESTIS

*Chowdhary N.D. **Khan A.R.

Abstract:

Of the 110 testicular tumors studied during last 12 years 13 cases (11.8 percent) were diagnosed as combined mixed germ cell tumors of testis. They included teratoma (Mature and Immature) with Yolk Sac tumor in three cases, teratoma (mature and immature) with Seminoma (typical) and Yolk Sac Tumor in 2 cases, Teratoma (mature and immature combined) with Seminoma (Typical) in 3 cases, Seminoma (Typical) with Yolk Sac Tumor in 3 cases, Seminoma (Typical) with embryonal carcinoma in one case. One case had five basic components which in order of proportion incouded seminoma (typical), embryonal carcinoma, choriocarcinoma, yolk sac tumor and teratoma (mature and immature).

Key words: Tumor,Testes.

Introduction:

In majority of germ cell tumors of testis, the tumor is comprised of more than one histological pattern. Spermatocytic seminoma is the only germ cell tumor of testis which tends to occur in pure form. Other forms may occur in combination. The detection of difference components and designation of combination has been source of confusion of tumor markers have made the identification of different components easier and have revealed more than one component in upto 62 percent of germ cell tumors. Various elements provide proper explanation for the bizarre clinical behavious of these tumors. They are also responsible for divergence between the histology of primary tumor and metastasis. A combination of embryonal carcinoma with teratoma has been labelled as teratocarcinoma for a long time.

Grossly these tumors are usually large with cystic areas. They may have areas of haemorrhage and necrosis. Yolk sac tumors and syncytiotrophoblasts are often over looked in histological sections, whereas embryonal carcinoma, teratoma are readily identifiable. We present 13 cases of combined germ cell tumors of testis seen over an 11 year period from 1983.

Materials and Methods:

The material for the present study was obtained from the biopsy records of the Pathology department, Government Medical College, Srinagar and a private Clinical Lab. (Khan's Clinical Lab.) from January, 1983 to December, 1994. A detailed study of there testicular tumors was carried out during the year 1993-94, to study the various histological patterns with reference to W.H.O classification. Fresh sections were cut from old blocks wherever necessary. Sections of the formalin fixed paraffin embeded material were stained by H&E, Periodic Acid Schiff, Alcian blue and Van Geisor and Reticulen stains.

Results:

The clinical presentation and the histological diagnoses of the patients are depicted in Table I. While majority of the patients presented with urilateral testicular swelling, one patient had bilateral testicular swelling and one patients presented with an abdominal mass.

Largest specimen measured 200x100x50 mm (case 1) which on cut section

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* Postgraduate Resident. ** Professor,
Pathology Department
Government Medical College, Srinagar.

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showed a tumor 190x90 mm with testis compressed on one side. Smallest specimen measured 60x40x30 mm with tumor measuring 30x30 mm (case 6). This patient had tumor in other testis also which measured 80x40 mm. Capsule was intact in all the cases and infiltration of cord was recorded in two cases. On cut section varying degree of haemorrhage was seen in all the cases. Ten tumors had associated necrosis which was extensive in six cases. Ten tumors exhibited microcytic area with mucinous looking surface in six of them. In 4 tumors cysts varying from 1 mm to 5 mm were alternating with solid lobulated areas. Cartilaginous surface was felt in three tumors.

The various histological patterns observed on light microscopy are depicted in Table I.

Discussion:

Collins and Pugh [1] in their series of 951 cases of testicular neoplasm observed 14 percent casas as combined germ cell tumors. Combinations they detected were those of seminoma with teratoma.

Wurster et al [2] commented upon occurance of foci of orchioblastoma in certain testicular teratoms, most of which morphologically fell into M.T.I (Malignant teratoma intermediate) group. The tissue they described had a pattern indistinguishable from Yolk Sac Tumor and authors reported an increased mortality in the first year among such patients.

Table I

Case No.	Age/Side	Clinical Presentation	Histological Diagnosis
01.	20 L	-L testicular swelling -Recent hemoptysis	T(M&I)+YST
02.	25 R UD	-Absence of R testis	St+YST
03.	25 R	-Painless Rt scrotal mass	YST+St
04.	40 R	-Rt testicular swelling	T(M&I)+St
05.	35	-Scrotal lump	T(M&I)+YT
06.	19 B.L	-Bilateral scrotal swelling	T(M&I)+YST (both-sides)
07.	25 R UD	-Absence of Rt testis -Abdominal Lump	T(M&I)+YST+St
08	30 R	-Rt testicular swelling	St+ELA
09	28 R	-Rt testicular swelling	St+YST
10.	30 L	-Rt testicular swelling	St+Teratoma
11.	40 L	-Rt testicular swelling	St+ELA+CCA+YST+(ML)
12.	20 R	-Rt testicular swelling	YST+St+T (M&I)
13.	20 R	-Rt testicular swelling	St+T (M&I)

R-	Right
L-	Left
UD-	Undescended
B.L.-	Bilateral
T-	Teratoma
(M&I)-	Mature & Immature
St-	Seminoma typical
YST-	Yolk Sac Tumor
ELA-	Embryonal carcinoma
CCA-	Choriocarcinoma

Talerman [3,4] described foci of Yolk Sac tumor in (MTI) MALIGNANT TERATOMA INTERMEDIATE which they thought were responsible for aggressive behaviour of these tumor.

We observed teratoma (mature and immature) with Yolk Sac Tumors in three cases, Teratoma (mature and immature) with

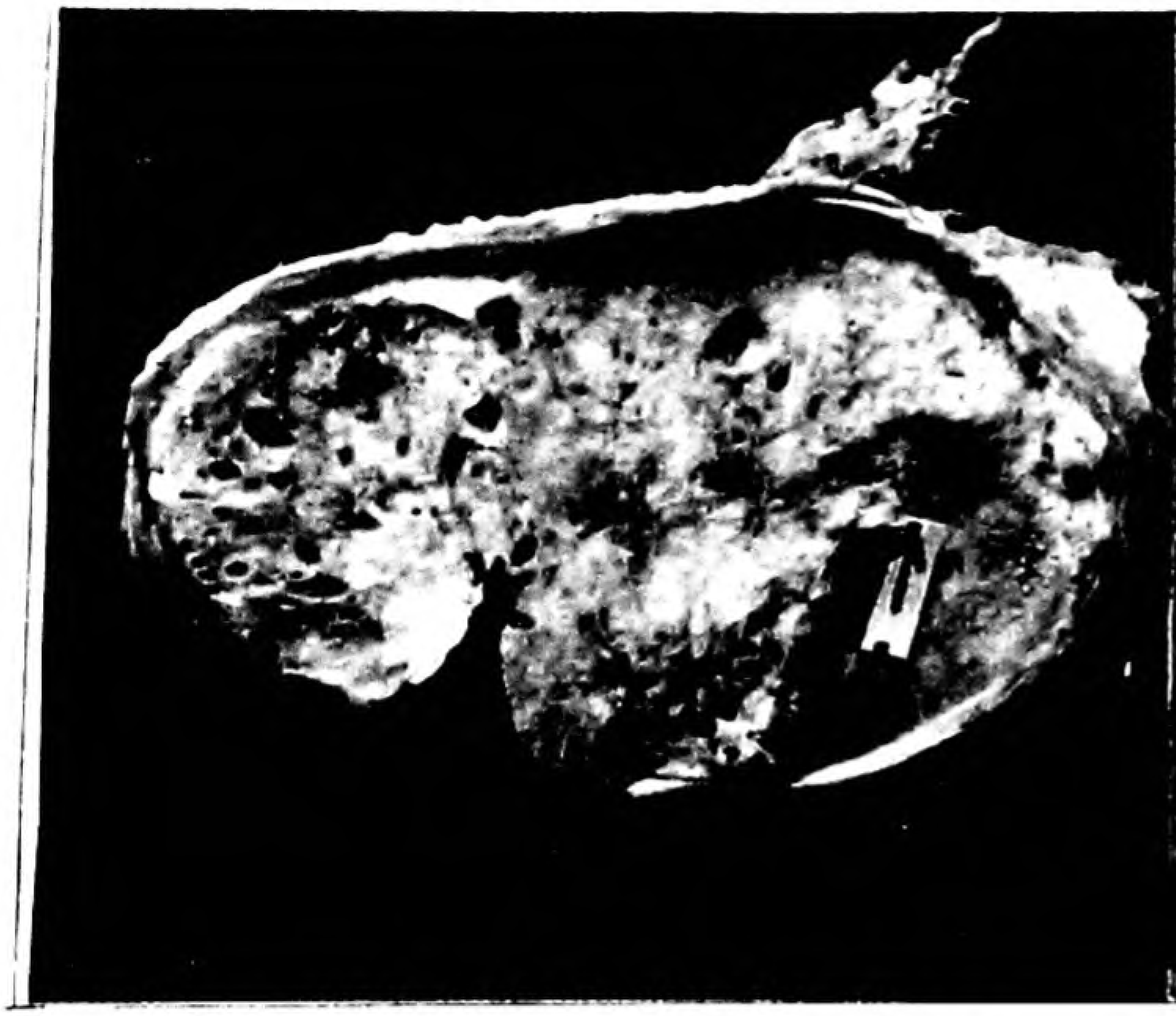

Fig I C/S of Combined Germ Cell Tumours

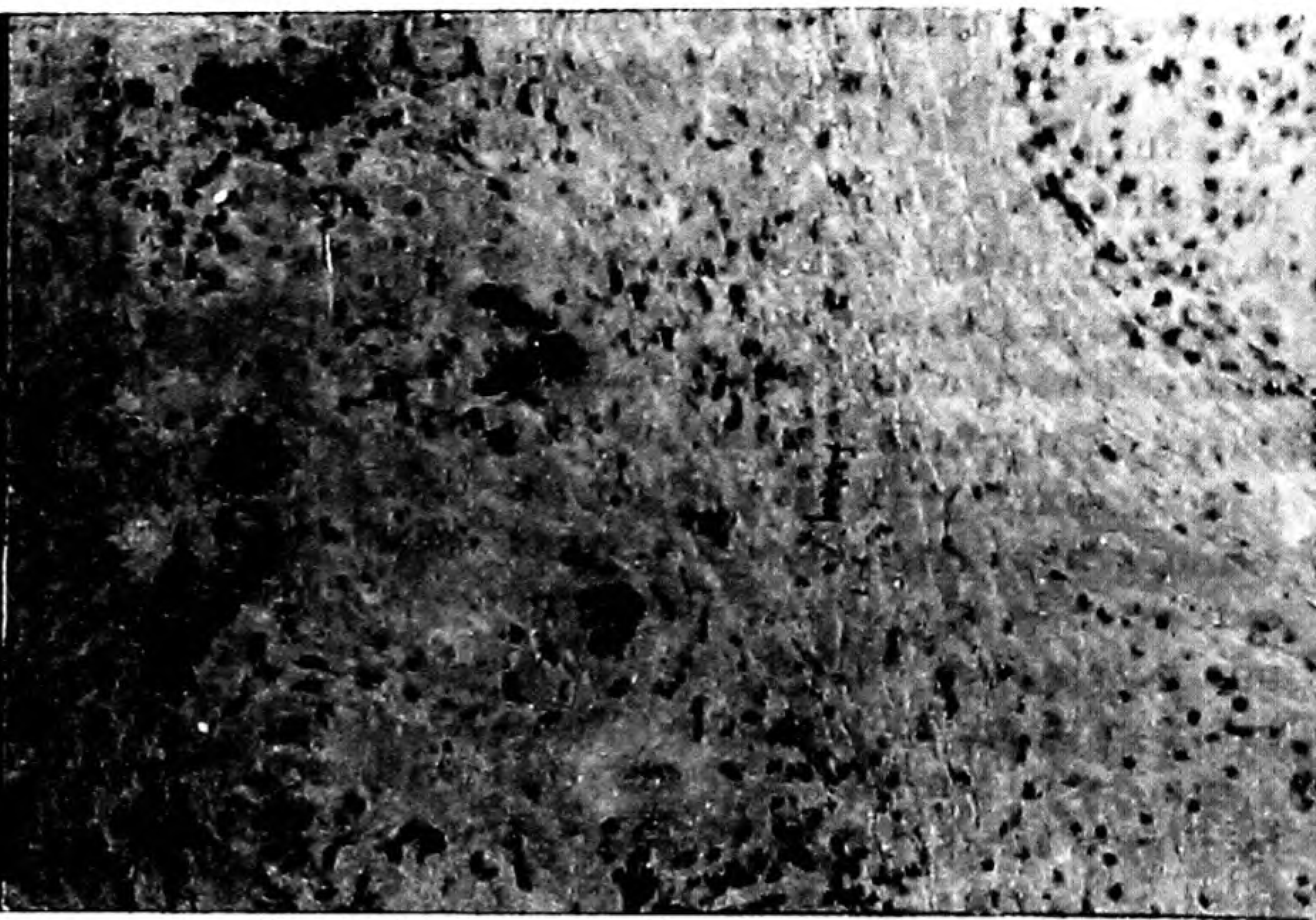

Fig II Microscropic Showing immature Cartilage, Stroma with immature nerve tubules (H & E x 400)

seminoma (typical) in 2 cases and in two more cases they had seminoma (typical) as third component; one case from formers (case No. 6) group had bilateral tumor in scrotal testis and one case of later group (Case No. 7) developed tumor in undescended testis at the age of 25 years. In three cases we observed a combination of seminoma (typical) with yolk sac tumor (pure) in equal proportion. There were three cases of seminoma with teratoma (mature and immature). Talerman [3,4] observed combinations of seminoma with yolk sac tumor in adults. One of three cases reported by author behaved as with cases of Pure Seminoma whereas two cases behaved agressively and author advocated unpredictivility of these tumors. Other investigators also have reported similar observations [2].

We observed a combination of seminoma with embryonal carcinoma in one case and one case carried the five basic components in it in fairly good proportion. They included in order of frequency : Seminoma (typical), Embryonal carcinoma Choriocarcinoma, Yolk Sac Tumor and Teratoma (mature and immature).

Mostofi [9] in a comprehensive review of 700 cases of testicular tumors reported most common association of teratoma with embryonal carcinoma (which occunted for 24 percent of testicular tumors), teratoma, embryonal carcinoma and seminoma in 6.4 percent, embryonal carcinoma and seminoma in 5 percent, seminoma and teratoma in 2 percent and embryonal carcinoma with choriocarcinoma in 1 percent. Further, Mostofi reported that 60 percent of testicular neoplasms had more than one histological pattern whereas in this study they comprised 12.1 percent. This disparity may be partly explained by inavailability of tumor markers in our study which is based purely on morphology.

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JK Practitioner 1996; 3:1 43-44

Case Reports

PAPILLON - LEFEVRE SYNDROME

*Qazi Masood, **Sheikh Manzoor

ABSTRACT

Papillon Lefevre syndrome (Syn - Palmoplanter Keratoderma with periodonitosis) is a rare genetically determined disorder of keratinization. We report a case of this syndrome.

CASE REPORT

A 10 year old female presented to us with few hyper-keratotic lesions over the hands and feet. These lesions were present from infancy and were progressing in size. The physical examination revealed reddened and thickened palms and soles. Lesions were extending to sides of feet and hands [Fig. 1]. The lesions showed scaling also.

There was loss of teeth [Fig. 2]. Hair and nails were normal, mucous membranes were also normal. X-ray skull was also normal. There was no tentorial calcification.

DISCUSSION

Papillon - Lefevre syndrome[1] (Syn - Palmoplanter keratoderma with periodontosis) is an inherited antosomal recessive disorder characterized by redness and thickening of the palms and soles; associated with periodontosis and a tendency to frequent pyogenic skin infection.

Redness and thickening of the palms and soles first appears between the age of 1 and 5 years and extend to the sides of the hands feet, and up the achilles tendon.

Sometime the changes on the knees and elbows are mistaken for psoriasis[2]. Associated hyperkeratosis causes an unpleasant odour[3]. The hair are usually normal but may be spasre. Frequent pyogenic infection of the skin occur[4]. Peridontosis resulting in severe ginginitis leads to the loss of teeth by the age of 4 or 5 years.

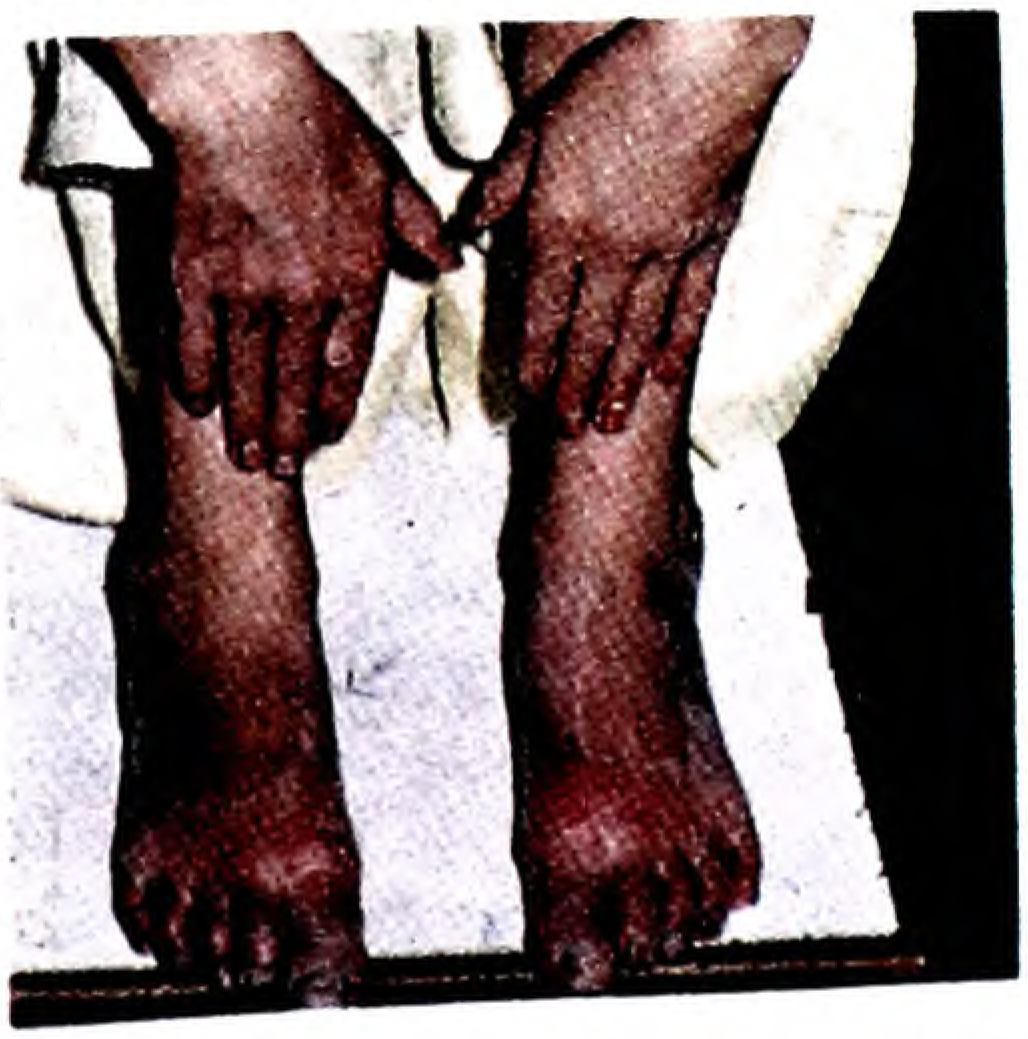

Fig !

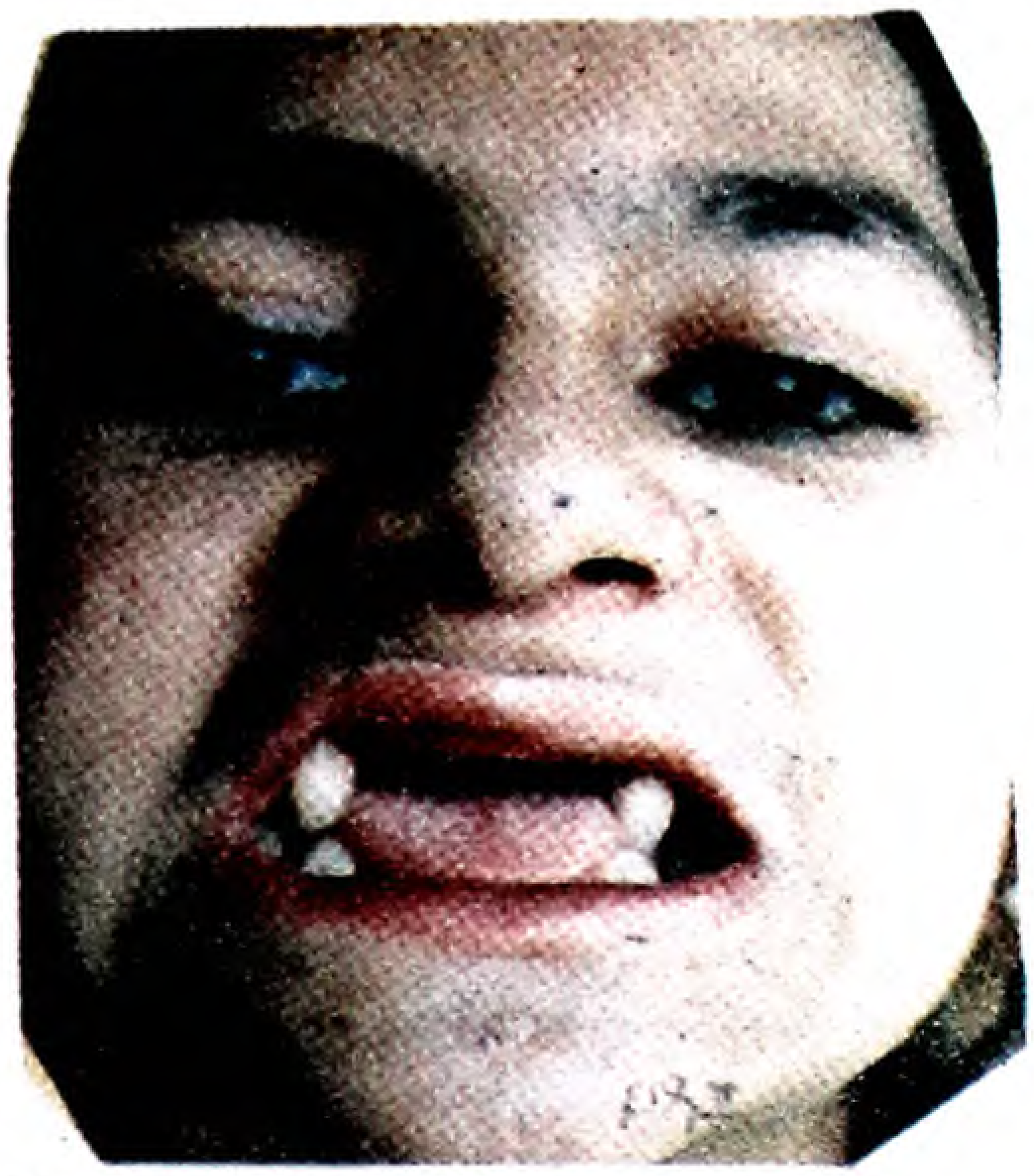

Fig II

Unless treated the permanent teeth may be lost in the same fashion. In our case report the erythema and scaling of the hands and feet

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* Assistant Professor
** Resident
Department of Dermatology
Govt. Medical College, Srinagar, Kashmir-190009.

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started during infancy, with loss of teeth temporary and permanent teeth. Her other system were normal. There was no dural calcification. Dural calcification has been noted in few cases. Before the advent of retinoids, dental clearance and antibiotic therapy was advised. But nowadays Etretinate, isotretinoin and acitretin have all been successful in improving the cutaneous eruption, lessening the gingival inflammation and in several cases saving the teeth.

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Case Report

JK Practitioner 1996; 3:1 44-46

SPINAL HYDATED CYST CAUSING CORD COMPRESSION

***Gh. Rasul Mir F.R.C.S. (U.K)**

ABSTRACT

A case report of the spinal cord compression due to hydated cyst is presented. The presentation mimics cord compression due to a dumb-bell tumour like neurofibromatosis. Operated two weeks after onset of cord compression, patient recovered fully and seen two years after surgery was well and symptom free.

KEY WORDS: SPINE, HYDATED CYST:

SPINAL HYDATED CYST CAUSING CORD COMPRESSION

Though hydated disease is not uncommon in India but it mainly involves liver and lungs, osseous involvement is rare and incidence of the spinal affection with paraplegia is about half of the total osseous involvement[1].

Very few cases of the disease have been presented so far in the literature. Woodlanc (1949) has reported six cases with paraplegia from Sydney. Berky (1954) from Turkey has reported two cases which improved after surgery. Aggarwal (1962) reported hydated cyst causing cauda equina syndrome[2], cases were also reported by Natrajan[3] (1974) and Bahulkar[4] (1984), others by 95.

Because of the rarity of the disease the diagnosis is often difficult before operation and it is, therefore, worth reporting such cases to remind ourselves of the existence of the disease so that a surprise finding at operation might not be misdealt which may lead to anaphylactic shock by spillage of the contents.

CASE REPORT:

Abdul Rahman, 28 years old male was admitted on 03.03.1994, in the Bone and Joint Surgery Hospital Barzulla, Srinagar with the complaint of:

01. Pain in the dorsal spine.
02. Inability to stand for the last 15 days.

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*Prof. Department of Orthopaedics
Govt. Medical College,
Srinagar, Kashmir, India

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About one year back the patient started with pain in the dorsal spine which was not radiated to any other part. The pain used to increase on coughing and sneezing. Fifteen days before admission the patient developed weakness in both lower limbs which was progressive in nature and then he was not able to stand. There was no history of trauma, cough or fever during this period. Bladder and bowel control were normal.

EXAMINATION:

General physical examination was negative. Chest and cardiovascular system were normal. Liver and spleen were not palpable.

Local Examination:

There was no swelling in the dorsal spine. Tenderness was present in the high dorsal spine and no deformity of dorsal spine.

Neurological Examination:

Higher functions were normal. Cranial nerves were normal. Both the upper limbs were normal.

Both lower limbs appeared wasted. Muscle tone was increases.

Muscle power was as follows:

Muscle	Right Side	Left Side
Hip:		
Flexors	3	3
Adductors	4	4
Gluteus maximus	3	3
Hip abductors	3	3
Knee:		
Quadriceps	3	3
Hamstring	3	3
Foot:		
Planter flexors	2	2
Invertors	1	1

Both superficial and deep sensations were intact.

Reflexes	Right Side	Left Side
Planter	Up going	Up going
Abdominal	-	-
Cremastic	-	-
Knee	Increased	Increased
Ankle	Increased	Increased.

INVESTIGATIONS

01. X-ray dorsal spine Erosion of the bodies of (D_2-D_4) on right side with enlargement of the foramina at these levels.

02. X-ray Chest: Normal.

03. Myelography: Showed obstruction to the flow of dye at the level of D-4, Fig. 1

04. Operative findings: On laminectomy D2, 3, 4 an extradrual hour glass shaped swelling was seen opposite to the D-2, 3, 4 vertebra and pressing the cord. While removing the cyst it got punctured and clear fluid came out from it. The sack was removed after separating it gently from the dural tube and was excised in total. The dura was not opened. Fig II.

FOLLOW-UP:

Immediately after operation he was put on Mebendizole which he continued for three months. Patient remained in hospital for about three weeks and he was able to more about with the help of a stick at the time of discharge. Six weeks later he was walking without any support and his muscle power in the lower limps was around grade 4 to 5. He was symptom free after two years after surgery.

DISCUSSION:

Hydated disease is very uncommon entity in Kashmir and spinal involvement, therefore, very rare. It is, therefore, seldom suspected clinically and pre-operative diagnosis nearly never made. The clinical picture mimic that of cord compression due to any other entity. X-ray picture also is not specific. In our case the patient presented with recent onset progressive paraplegia and x-ray showed enlargement of intervertebral foramins between D-2 to D-4 and erosion right side of vertebra. This is similar to be seen in dumb-bell tumour of neurofibromatosis. If decompression is done early the recovery is generally full. However, caution has to be exerted that entire wall of the cyst must come

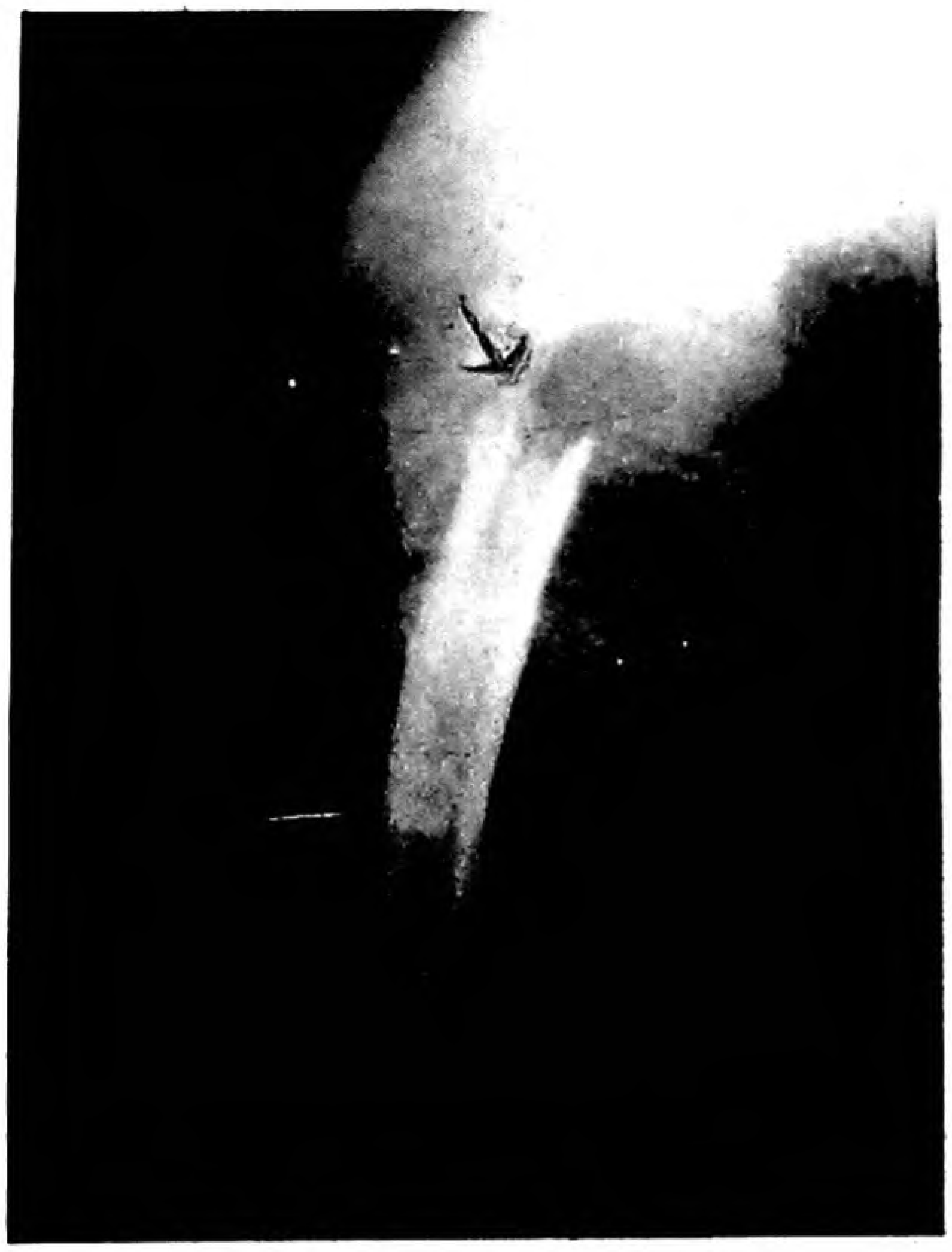

Fig I Myelogram showing spinal block.

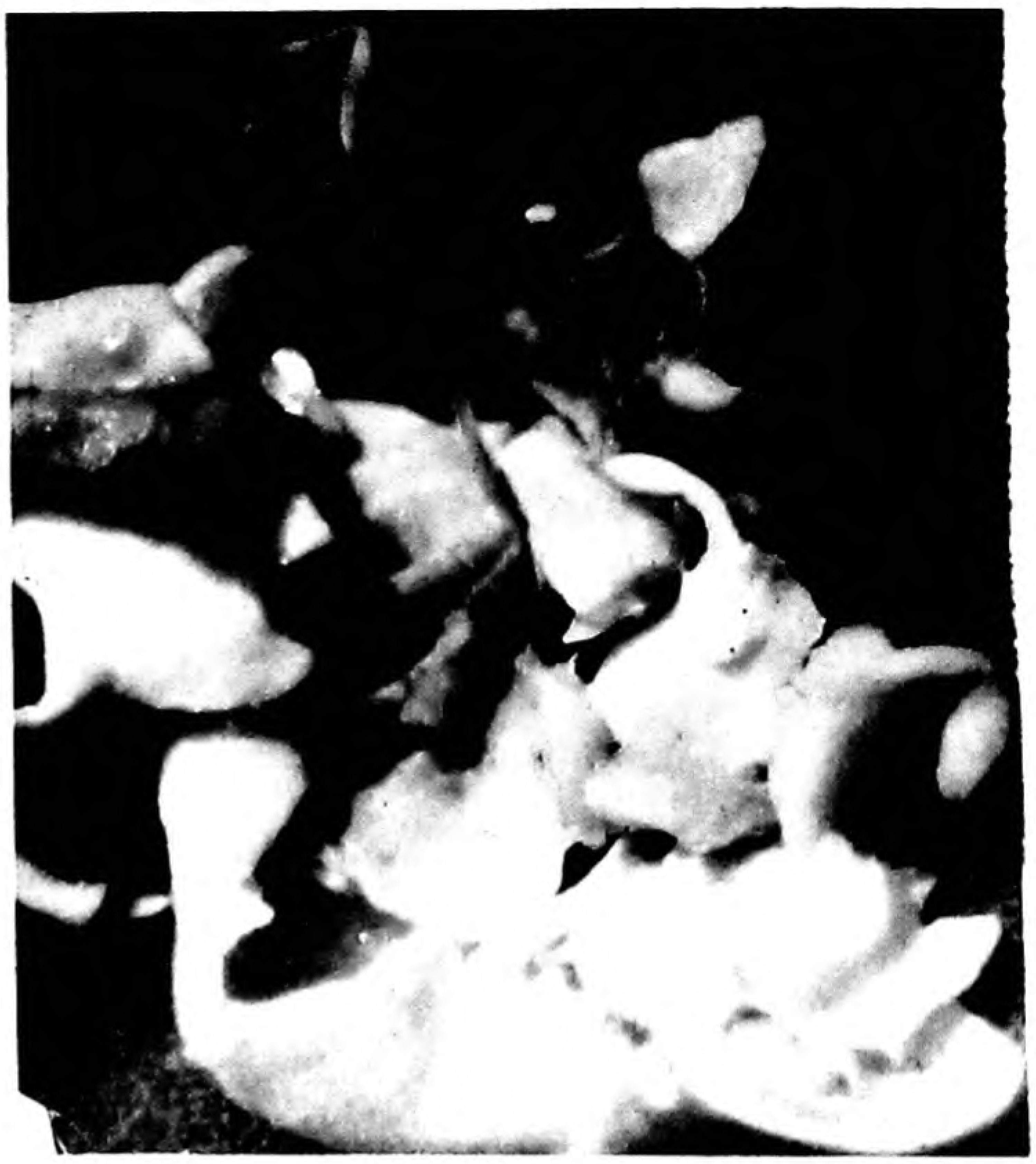

Fig II Hydated Cyst Spine (Specimen)

out and subarachnoid space must not open as the wall of the cyst is adherent to the dura and while peeling it dura may tear. Leakage of contents of cyst may produce anaphylactic shock. Treatment with Mebendazole for three months must be instituted after the diagnosis to ensure complete cure of the disease.

Address:
Prof. G. R. Mir FRCS.
Hospital for Bone & Joint Surgery
Barzulla, Srinagar, Kashmir, India
Telephone: 30129, 30149

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Case Reports

PNEUMOCEPHALUS FOLLOWING NASAL POLYPECTOMY

***Nazir A. Bhat**

Pneumocephalus following intranasal polypectomy is very rare. We present a case which we initially though was a CVA (cerebo vascular accident).

Keywords: CVA; Nasal Polyp; Pneumocephalus.

Introduction:

Pneumocephalus is defined as a collection of air or gas within the skull. The air may be epidural, subdural, subarachnoid, intraventricular or intercerebral. Most common causes of pneumocephalus, in decreasing order of frequency, are neoplasm, infections and surgical intervention [1]. Pneumocephalus following nasal polypectomy is very rare. In a review of the literature from 1884 to 1967, Markham found only one reported case. Since then, only three more cases have been reported [2-4]. In two reports reviewing more than 2000 patients operated on, no such complication occurred [5-6]. Herewith we report one additional case.

Case Report:

An 82 year old hypertensive and asthmatic lady presented to our outpatients with a history of right sided nasal obstruction of five months duration. Examination of the nose revealed a single polyp in the right nostril. The rest of the ear, nose and throat examination was unremarkable. Intranasal polypectomy was done under general anaesthesia. The following morning the nasal pack was removed. Bruising was noticed below the right intraorbital margin. The same evening the patient was found on the floor by the nursing staff on the side of the bed.

On enquiring the patient said that she had a frontal headache all day and she was trying to get out of bed because she felt nauseated. She felt dizzy and landed on the floor.

On examination she was found to be orientated in time, space and place and was haemodynamically stable and afebrile. She had left 7th nerve LMN palsy and hemiparesis of the left leg. Our first impression was that she had probably CVA (cerebro vascular accident). A CT Scan was done to find whether the CVA was due to Thrombo-embolism or Intra cerebral haemorrhage, but it revealed extensive air within the subdural and subarachnoid spaces (Fig. I). No CSF rhinorrhoea was noted.

She was put on prophylactic antibiotics and was to be rescanned in a weeks time. After one week the patient had no neuro-deficit in the limbs and the left 7th nerve had conmpletely recovered. The repeat CT scan was done on the 11th post-operative day and revealed almost complete absorption of air with no SCF rhinorrhoea (Fig II).

The patient was found fit and discharged on the 20th post-operative day. She was reviewed in the clinic after 4 weeks and was found in a good state of health. The pathological examination showed an inflammatory polyp.

Discussion:

Lucket [7] was the first to demonstrate intra-cranial air radiographically. Markham [1], reported that surgical procedures (mainly neurosurgical) were responsible for 3.7% of 284 cases of pneumocephalus. Headache was

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*Registrar E.N.T. Deptt.
10 - Torrant Walk
Walsgrave Conventy (UK)

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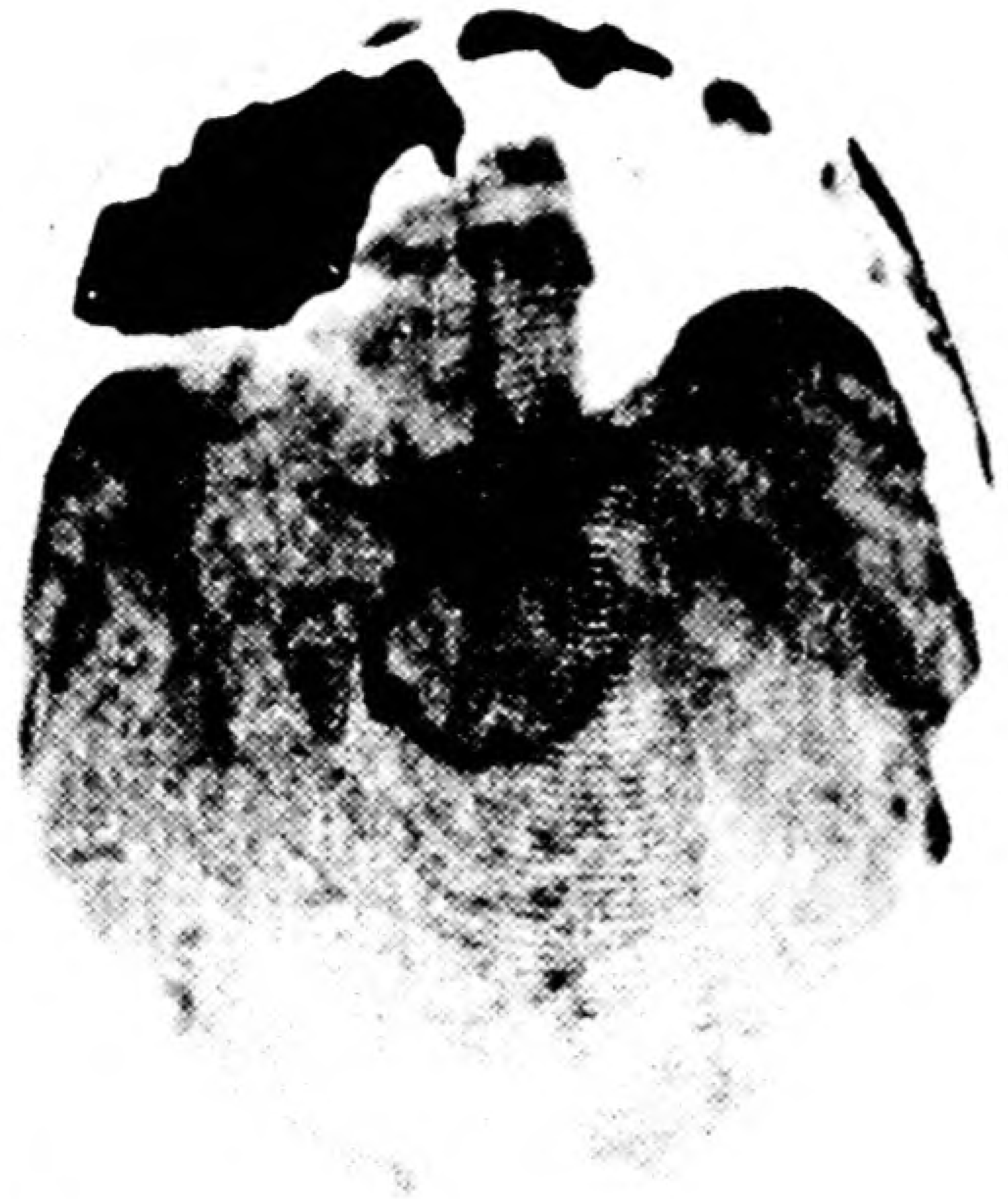

Fig I

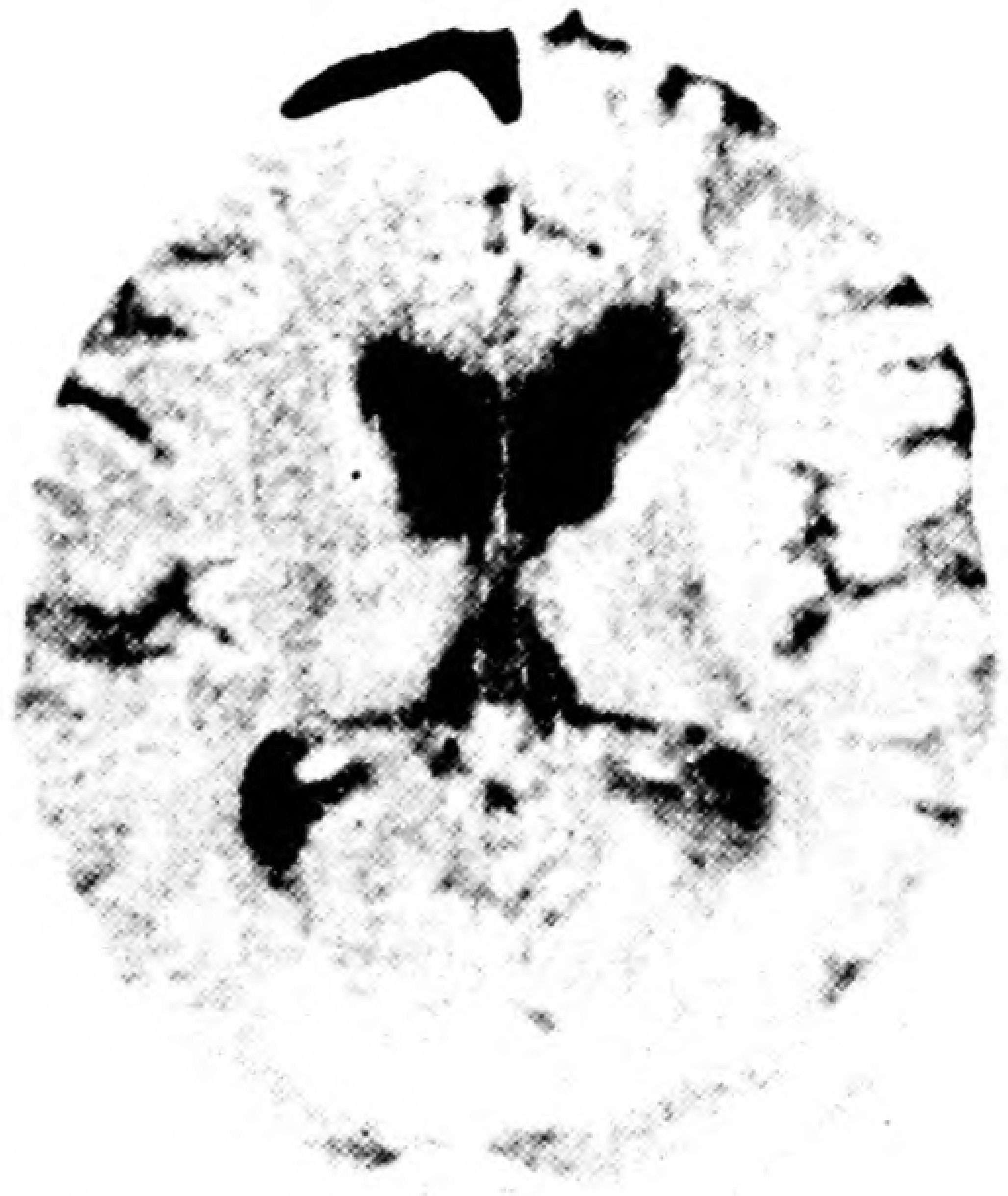

Fig II

the most common symptom. CSF rhinorrhoea occured in only one third of patients.

Two mechanisms have been postulated in the development of pneumocephalus. The "ball valve" mechanism requires that air under high pressure will be forced intracranially. A prerequisite is a defect in the skull bases and/ or dura. Coughing, sneezing or other causes of increased intrapharygeal pressure may force air into the skull and its exit is prevented by sealing of the defect tiwh dura or brain tissue, or even by blood clots and edematous nasal mucosa. The "inverted bottle" mehcnism is the result of excessive CSF leakage causing slightly negative intra-cranial pressure, and as a result, air is driven intracranially. It is reasonable to assume that the ball valve mechanism acted in our patient, since in this condition air may enter the skull without reciprocal loss of CSF, and indeed, at no stage during hospitalization did CSF rhinorrhoea occur.

Conclusion:

A diagnosis of Pneumocephalus should be suspected in a patient following nasal or paranasal surgery who has persistent headaches with or without CSF leakage. Conservative treatment with prophylactic antibiotics is preferrable in traumatic type. Surgical treatment is indicated in cases of continuous CSF rhinorrhoea, recurrent meningitis or signs of increased intracranial pressur due to air accumulation.

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JK Practitioner 1996; 3:1 49-52

Practitioners Column

CLINICAL MANIFESTATIONS OF AIDS

*G.H.Hajini

Viral replication and progressive immunologic impairment occurs throughout the course of HIV infection, so that the disease has a progressive course even during clinical latency. On the basis of the degree of immunosupression HIV disease can be divided into:

a. Early stage with CD4+T cell count of greater than 500/ml.
b. Intermediate stage of CD+T cell count of between 200-500/ml.
c. Advanced stage of CD4+T cell count less than 200/ml.

Most of the opportunistic infections and true malignancies occur in the advanced stage, where as neurological disease and Kaposis sarcoma are not strictly related to the degree of immunosupression. Though there are no recognized HLA associations with HIV infection, GLA-B35 is however reported to be associated with accelerated progression of AIDS.

Skin manifestations:

Many patients of AIDS have cutaneous manifestations. Since some of these lesions particularly in combination with other HIV related signs and symptoms are fairly specific for AIDS, their recognition may lead to its early diagnosis. Other skin lesions are often nonspecific but these may be manifestation of systemic disease. Generalized prugigo is found in some 20% of patients of HIV infection in Africa. Once appeared these continue throughout the course of the disease. These are symmetrically distributed mostly on limbs especially on extensors. Their etiology is unknown and are non-specific histologically. In one study 87% patients of prurigo in out patients were HIV antibody positive. Skin manifestations include:

a. Neoplasia:
(1) Kaposis sarcoma (34% of AIDS patients) (2) Lymphoma (3) Intraepithelial cancer (4) Intraoral squamous cell Ca. (5) Squamous and basal cells Ca (6) Malignant melanoma.

b. Infections:
(1) Herpes zoster, meningitis, encephalitis (2) Harpes virus infection (3) Dermatomycosis (about 30% patients), keratoderma blenorrhagica like picture in soles (4) Candidiasis angular stomatitis, onychia (5) Epstein Barr virus infection (6) Cryptococcosis (7) Histoplasmosis (8) Molluscum contagiosum (9) Condylomata accuminata (10) Chancroid (11) Syphilis (frequent co-existences) (12) L.G.V (13) Impetigo (14) Folliculitis (15) Furunculosis (16) Tuberculosis (17) Pyomyositis (18) Demodicidosis (folliculitis to demodex folliculorum) (19) Bacillary (epitheloid) angiomatosis (20) Bacterial angular stomatitis (21) Pityriasis versicolor (22) Pityriase rosacea (23) Scabies (24) Disseminated vaccinia (25) Multiple papules caused by acanthamoeba castellani (26) Sporothrix schenkii infection (27) Fleshy growths in external auditory canal caused by pneumocystitis carinii.

Others:
(1) Pruritic popular dermatitis (2) Seborrheic dermatitis (3) Drug eruptions (sulphonamides

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* Prof. & Head Dermotology, STD & Leprosy
Medical College Srinagar, Kashmir
Pin 190 009

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cause eruption in about 50% of cases) (4) Vasculitis (5) Xeroderma (6) Psoriasis (7) Granuloma annular (8) Thrombocytopenic purpura (9) Telangiectasia (10) Hyperpigmentation (11) Dry atrophic skin (12) Hair changes like: alopecia areata, Diffuse alopecia, Premature graying, vertical thinning, localized loss around scalp margins, Elongation of eye lashes (13) Increased insect bite reactions (14) Hyperplastic painful keratotic lesions (15) Erythroderma (16) Erythema elevation diutenum (17) Porphyria cutanea tarda (18) Eruptive dysplastic nevi (19) Glucagan induced keratoderma (20) Grover's acantholytic dermatosis (21) Pitted keratolysis (22) Angiolipomas (23) Photosensitive eruptions (24) Dupytrens (25) Kawasaki disease (26) Panniculitis (27) Lichen planus.

Oral lesions: include:

(i) Herpes simplex (ii) Thrush (iii) Hairy leukoplakia (iv) Persistent aphthous type ulcers (v) Zoster (vi) Warts (vii) Gingivitis (viii) Perioeontal disease (ix) Xerostomia (x) Parotid swelling (xi) Kaposis sarcoma. Thrush and hairy leukoplakia are usually indicative of advanced immunological decline generally occurring in patients with less than 300 CD4+T cells-ml. In one study 59% patients with oral candidiasis developed AIDS over next year.

Neurological manifestations:

Nervous system involvement accounts for a high degree of morbidity in a good number of patients with HIV infection Neurologic involvement can occur by (a) Primary HIV infection as i. Aseptic meningitis ii. AIDS dementia complex iii. Myelopathy iv. Peripheral neuropathy v. Myopathy. (b) Secondary to opportunistic infections like Toxoplasmosis, Cryptococcosis, multifocal leukoencephalopathy, CMV, HTLVI, tuberculosis, syphilis, papovavirus, Herpes virus candid albicans etc. (c) Neoplasms i. Primary CNS lymphoma ii. Kaposis sarcoma. Pathology may be inflammatory, demyelinating or degenerative in nature. Damage to CNS may be the direct result of viral infection of CNS macrophages, or glial cells or secondary to the release of toxic cytokines where as most of the HIV infected patients will have some neurological problem during the course of their disease, only AIDS dementia complex is considered as AIDS defining illness. CSF findings are abnormal in about 90% even at the asymptomatic stage of HIV infection in the form of pleocytosis, elevated proteins, presence of virus and anti HIV antibodies.

Aseptic meningitis is suggested to be an immunologically mediated disease and may be seen through all but the very late stages of the infection with clinical features varying from mild headache to frank encephalitis and CSF showing pleocytosis and elevated proteins. Condition resolves spontaneously within 2-4 weeks, but may persist as a chronic problem.

AIDS dementia complex usually occurs late in the course of infection. Dementia is a major feature. Patient may also have motor and behavioral abnormalities including unsteady gait, poor balance, rapid alternative movements, apathy, lack of initiative. AIDS dementia is the initial AIDS defining illness in about 10% cases with HIV infection and thus rarely occurs prior to clinical evidence of immunodeficiency and eventually develops in 2/3rd of patients and is regarded to be due to direct affect of HIV infection of CNS.

Spinal cord disease is seen in about 20% patients of AIDS and include (a) vascular myelopathy (b) pure sensory ataxia due to dorsal column involvement (c) Parasthesias and dysesthesias of lower limbs.

Peripheral neuropathy: is a common occurrence during all stages of AIDS illness and appears in any of the following forms (a) mononeuritis multiplex (b) distal symmetrical painful polyneuropathy seen in 30-40% patients (c) acute inflammatory demyelinating polyneuropathy.

Myopathy: It may also complicate the course of HIV infection and may be due to HIV infection, zidovudine toxicity or part of generalized waisting syndrome.

Autonomic nervous system involvement: Orthostatic hypotension, cardiorespiratory arrest related to autonomic neuropathy is reported in HIV infected patients.

G. I. manifestations:

(a) Persistent diarrhoea is generally of unknown etiology but may be due to cryptosporidiosis, isosporiosis, other enteric micro organisms.

(b) Dysphagia due to candidal eosophagitis in over 90% cases, Herpes esophagitis, eosophagitis due to cytomegalo virus or other ulcerations of unknown etiology.

(c) Colitis: Occasionally due to cytomegalo virus or kaposis sarcoma but generally of unknown cause.

(d) Parianal discomfort due herpes virus proctitis or ulcerations.

Anogenital infections and tumours:

Sexually transmitted diseases are often seen in HIV infected adults. Ulcerations in this region could be herpetic, syphilitic or chancroidal. Other lesions include warts and increased prevâlence of cervical dysplasia. HIV associated cervical and anal cancers are distinctly aggressive.

Respiratory manifestation:

(a) Persistent cough, dyspnea, cynosis, tachypnoea caused by tuberculosis, pneunocystis carinii infection, Bacterial pneumonia atypical mycobacteria, candida, cytomegalovirus, cryptococcus neoformans, Histoplasma capsulatum, Legionella pneumophilia, Nocardia asteroides, Lymphoid interstitial pneumonitis, Kaposis sarcoma.

(b) Haemoptysis and pleural effusion due to tuberculosis for kaposis sarcoma.

Lymphadenopathy:

Persistent generalized lymphadenopathy (PGL) is defined as an enlargement of lymph nodes of more than 1 cm diameter in 2 or more extrainguinal sties of east 3 months duration in absence of any known cause of lymph adenopathy, is often the earliest sign of HIV infection following the acute syndrome. Inguinal nodes should be disregarded for the diagnosis of PGL. More than 50% patients of HIV infection develops PGL within months of infection and may persist unchanged for several years. Lymphnodes may regress or disappear when AIDS stage is reached. In an Africa study lymphnodes were detectable in 10-20% of full blown AIDS patients. Splenomegal is present in about 10-30% of patients of PGL. Although lymphadenopathy is not associated with any increased likelihood of developing AIDS, paradoxically a loss of lymphadenopathy of a decrease in the lymphnode size may serve as a poor prognostic marker and indicative of disease progression.

Haematological manifestations:

Anemia is often present particularly in advanced stages of HIV infection. Though in most cases cause remain unknown, in some it is due to infection, malignancies, malnutrition and toxic drug treatments.

Thrombocytopenia may be an early consequence of HIV infection. In AIDS patients notable are leukopenia, lymphopenia, Bone-marrow hyperplasia, peripheral cytopenia and clinically cutaneous/oral petechiae and ecchymosis.

Renal manifestations:

Nephropathy in AIDS in characterized by golmemlosclerosis and proteinuria, manifesting clinically as nephrotic syndrome with or without renal insufficiency. There is rapid deterioration of renal function. Nephrotoxic drugs, sepsis, hypoxia, hypotension and dehydration may contribute to acute renal failure in AIDS patients.

Cardiac manifestations:

Tuberculosis pericarditis is a common

cause of pericardial effusion in HIV infection.

Adrenal insufficiency:

AIDS patients may sometimes develop adrenal insufficiency. Most cases of necrotizing. Adrenalitis are due to CMV infection. Adrenal glands may also get infected by mycobacteria and cryptococci, Kaposis sarcoma may involve adrenal glands.

Hepatic disease:

Liver is involved frequently in AIDS patients. Opportunistic infections and malignancies account for 33-78% of post-mortem lesions, there is however, no pathognomic lesion in liver due to HIV infection.

Joint manifestations:

Reiters syndrome associated with HIV infection and subacute oligoarthrtic syndrome have been reported. HIV has been isolated from synovial fluid.

Reproductive system manifestations:

Ammenorrhoea is reported in 40% of women with AIDS and there is often associated weight loss. Hypogonadism in HIV patients is also reported.

Eye manifestations:

Retinal abnormalities are common in AIDS patients, with usually no visual deficiency. Fundoscopy shows soft exudates which are self limiting. Commonest cause of loss of visual acuity which occurs late in the disease process is due to cytomegalovirus (CNV) retinitis vascular occlusion and haemorrhages may be present. Ocular toxoplasmosis, zoster infection and kaposis sarcoma are other occasional lessions.

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JK Practitioner 1996; 3:1 53-55

Practitioners Column

Haematuria - Do we manage it correctly:

*Gulam Jeelani; **Mumtaz-Din

Introduction:

Haematuria i.e. blod in urine is a very common urological symptom usually quite frightening to the patient if observed grossly. Haematuria must be distinguished from other causes of reddish discolouration of the urine which include haemoglobinuria, porphyria, the ingestion of certain drugs, red beet or purple cabbage, colouring agents used in bakery like rhodamine-B. As such it is not a disease by itsaelf but a symptom/sign of underlying disease in the genitourinary tract. Essentially it can be gross or microcopic, painful or painless, perse, or associated with other urological symptoms, throughout the act of micturition, terminal or initial and continuous or intermittent.

By and large patients with gross haematuria report to the doctor early while as those with microscopic haematuria is picked up by the practitioner during routine microscopy while treating some other non-urological ailment. In both situations a large number of cases are not evaluated either fully or properly and often there is a time gap between the initial consultation of the patient and the subsequent urological referral.

When a patient of haematuria whether gross or microscopic is seen by the practitioner it is imperative to evaluate these patients on a difinitive guideline so that if a significant cause for this symptom is detected.

Therefore this article is being written with practitioner in view who has a chance and responsibility to see and evaluate the patient of haematuria initially with a hope that these patients who may be barbouring with very serious disorders as the cause of haematuria are managed properly.

Causes of Haematuria:

The causes of haematuria can be conveniently divided into two groups:

A. Surgical causes of haematuria.

B. Haematuria due to Medical Renal disease.

A. Surgical cause of heamaturia:
- I Congenital
- II Acquired.

I Congenital:

- a. Congenital polycystic disease of kidneys.
- b. Medullary sponge kidney.
- c. Congenital obstructive uropathies with secondary infection.
- d. Malpositioned kidenys i.e. pelvic, ectopic, horse-shoe and malrotated.
- e. Congenital renal Arterio-Venous malformation.
- f. Hydronephrosis
- g. Solitary renal cyst

II Acquired:
- a. Traumatic: -Blunt injuries
  - -Gun shot wounds
  - -Insturmentation
  - -Stab and balst injuries
  - -Iatrogenic injuries.

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* Associate Professor Surgery (Urology)
** Lecturer Deptt. of Surgery
Medical College Srinagar, Kashmir - 190 009.

b. Inflammatory: Acute
urinary tractinfection
(of upper or lower urinary tract).

Chronic urine infection/Inflammations like tuberculosis, Bilharziasis, chronic nonspecific cystitis, interstitial nephritis (Papillary necrosis).

c. Calculous disease: Stones any where in urinary tract are important causes of haematuria in every age group.

d. Neoplasma:
  i) Benign prostatic Hyperplasia, cysts
  ii) Haemangionas
    Wilm's tumour
    Renal cell carconoma of kidenys
    Transitional cell carcinoma of bladder Carcinoma of prostate •

e. Miscellaneous: Foreign bodies in bladder urinary fistulas.

B. Medical causes of Hematuria:

1. Acute glomerulonephritis,
   IgA Nephropathy,
   Good pasteurs syndrome.
2. Blood dyscrasias : Purpura
   Sickle cell trait
3. Anticoagulant drugs
4. Analgesic Nephropathy
5. Collagen diseases
6. Subacute bacterial endocarditis
7. Systemic lupus erythematosus
8. Polyarteritis nodosa
9. Jogger's haematuria

Management of Haematuria:

I. History taking: When a case of haematuria is seen by a practitioner a proper history keeping in mind the duration, whether haematuria is gross or microscopic, painful or painless, initial, throughout or terminal, whether associated with other symptoms of pain in the genitourinary tract or symptoms of lower tract irritiation like dysuria frequency, urgency are important to be noted.

Generally paroxysmal, painless haematuria of long standing duration are indicative of a tumour in the urinary tract. Similarly painful haematuria which is usually associated with other irritative symptoms of lower tract like dysuria, frequency urgency are indicative of benign conditions like stone, cystitis, BPH etc. Similarly haematuria occuring before begining of micturition is indicative of urethral pathology like calculous, tumour, inflammations. Haematuria occuring throughout the act of micturition has usuallyits origin from bladder or kidneys. Terminal haematuria is often a symptom of bladder neck pathology or trigone.

Relevant history of blood dyscrasias, puffiness of face edema, oliguria may be pointers to Medical renal disease.

II. Examination of genitourinary tract: This should include examination of abdomen especially flanks to rule out or confirm the presence of a renal swelling which could be due to cyst, hydronephrosis, tumour of kideny suprapubic examination should confirm or rule out presence of a full bladder due to obstructive uropathies of lower tract which may be the cause of haematuria. External genitalia should be examined in detail which may confirm the presence of tubercular or nonspecific epididymorchitis. Lastly it cannot be too impressed that digital rectal examination (DRE) forms important part of examination in every case. Conditions like BPH, CA prostate and tubercular pathology in the genital tract can have specific findings on rectal examination. Finally findings like edema of feet, puffiness of face, Hypertension, anaemia and features of uraemia may be indicative of medical renal disease as the cause of haematuria.

Investigations:

1. Blood examination: for coagulation profile, blood urea, serum creatinine, serum electrolytes will help to establish the possibility of medical renal diease as cause of haematuria.

2. Urine Analysis: Urine analysis forms an initial and mainstay of the diagnositc work up. This is the area where certain important precautions in methodology and interpretation of the results are needed. Urine analysis must be done by morning, midstream, uncentrifuged sample collected in a sterile container and set to the laboratory immediately for evaluation and patient or a normal fluid intake. While interpreting it is important to know what is normal RBC excertion per high power field (HPF) of a centrifuged sample of urine. Upto 1 REC/HPF in males in some of the urine analysis (not persistent) is considered to be normal, in females however between 1-3 REC/HPF is considered non significant providing not persistent. In female patients it is important to take the menstrual history and refrain from interpreting a urine analysis during and at least for three days upto stopage of cycle. Presence of micropyuria in association with haematuria is usually indicative of urinary tract infection which must be confirmed by urine culture and sensitivity. Similarly presence of granular/hyalive casts are pointers to glomerulonephritis of various types. Rarely pneumaturia and faecaluria can be indicative of a urinary fistula. Urine examination can also be helpful in diagnosis of conditions like bladder tumour when malignant cells can be seen and in cases of tuberculosis where AFB can be isolated and stained from urine samples.

3. KUB/IVU: KUB will delineate renal outline and radiopaque stones in genitourinary tract will be picked up in majority of the cases. IVU will assess the functional status of kidneys besides picking up most of the pathologies ingenitourinary tract, however its role being invasive and least cost effective compared to ultrasound has become more and more limited.

4. Ultrasonography of genitourinary tract: Majority fo the pathologies in G.U tract are easily diagnosed by U/S which may be the cause of haematuria.

5. Cystoscopy: Cystoscopy can easily diagnose pathologies in bladder and urethra like stones, tumours, infections, fistulas, and prostatic pathologies.

6. Contrast enhanced CT scan and angiography are rarely needed in cases of difficult diagnosis, renal infuries, vascular malformations etc. as the cause of haematuria.

7. Prostatic biopsy may be indicated in some cases with abnormal findings on digital rectal examination to diagnose prostatic CA and tuberculosis of genitourinary tract.

Treatment:

Haematuria being a symptom complex of an underlying urological disease the treatment will depend on the underlying causative pathology.

It is imperative to realise that once the practitioner who has seen and evaluated the patient of haematuria initially must be clear whether he is dealing with medical renal disease as a cause of haematuria when perhaps he can treat the patient himself or seek the proper consultation from a physician/Nephrologist or else whether the cause of haematuria is surgical when he sould seek a urological consultation/referral to a proper centre.

CME Programme

JK Practitioner 1996; 3:1 56-59

TUBERCULOSIS AS WE FACE IT IN KASHMIR !

* M. Y. Bhat, MD,

The discovery of tubercle bacillus (the causative organism of tuberculosis) by Robert Koch in 1882 was the started point for many further advances, both practical and theoretical. With the development of many diagnostic techniques and the advent of effective chemotherapy, the incidence of tuberculosis was showing a declining trend till the recent past, especially in the developed countries. However, with a rising incidence of HIV infection, the disease has re-emerged with a bang. The clinical experience has revealed that the Kashmir valley is almost flooded with tuberculosis. Keeping in view such a high incidence, it seemed to be of interest to conduct a seminar on various aspects of tuberculosis at Government Medical College, Srinagar on 11th October, 1995. In his introductory address, Prof. Mohammad Yousuf gave a brief know-how about the measures taken in combating the disease and the work done so far by the Department of Medicine as also the future challenge in this regard.

CHALLENGE OF TUBERCULOSIS

Tuberculosis is back, is the latest about tuberculosis. There is steadily rising trend in tuberculosis, even from countries of the Word reporting no cases of tuberculosis since 1962. The actual number of tuberculosis cases from 1987-1993 from an American City exceeded the predicted number of over 51,000. Further epidemiological data show that there are 30 million active cases of the disease in the world, out of which 3 million die every year[1]. The rising trend in developed world may be due to rising incidence of HIV infection, but under our setting this factor may not be important at this time. However, we need to update our epidemiological data.

Data obtained from chest disease Hospital, Srinagar reveals that about 950 cases of pulmonary tuberculosis have been admitted during 1994-95, which is in addition to a large number being treated on an out-patient basis[2].

Table Showing the total number of cases seen in OPD & the number admitted (with special reference to tuberculosis cases) at CD Hospital,Srinagar 1994-1995.

MONTH/ YEAR	NO. OF PATIENTS SEEN IN OPD	NO. OF PATIENTS ADMITTED	NO. OF TUBER-CULOSIS CASES
04/1994	6174	496	118
05/1994	3443	429	74
06/1994	2748	453	82
07/1994	4763	309	134
08/1994	8108	386	48
09/1994	4198	289	98
10/1994	4436	394	99
11/1994	3274	381	97
12/1994	3986	383	47
01/1995	3930	375	38
02/1995	3906	384	56
03/1995	5646	512	48
Total	54612	4791	939

HOW HAVE WE TAKEN THE CHALLENGE

The challenge of this disease is not new to us. Even before the advent of our Medical College, this problem did receive attention. The basic diagnostic (X-ray and sputum examination) and treatment facilities have been available for many years. We had a tuberculosis sanatorium at Tangmerg. However, after Medical College came into being, the problem received greater attention and various basic research programmes were set up by the Department of Medical of this College. The work was done on this subject under the supervision of several teachers of our Deptt. Many postgraduate scholars undertook this work. Important observations made out of these studies are mentioned briefly in a following few headings.

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* Prof. Department of Medicine,
Govt. Medical College Associated SMHS Hospital,
Srinagar Kashmir (190010) India.

CLINICAL AND SOME DIAGNOSTIC ASPECTS

1. Majority of the patients have symptoms of tuberculosis on an average for six months before the disease is diagnosed, suggesting a delay in diagnosis.
2. Montoux test is positive in 96% of our cases.
3. 74% of our cases have advanced disease at the time of initial diagnosis, suggesting delayed reporting of cases.
4. ESR is raised in 92% of cases and correlates with the severity of the disease.
5. Authors suggest that high index of suspicion should be kept on cases with suggestive symptoms to make an early diagnosis[3].

SERUM IMMUNOGLOBULIN PROFILE

The mean IgG and IgA levels were significantly increased ($p < 0.001$) in the patient group as compared to controls. The mean serum IgA significantly correlated with the absolute lymphocyte count ($p < 0.05$) and the duration of illness ($p < 0.001$). The mean IgA level showed a significant increase ($p < 0.001$) in patients with poor nutritional status[4,5].

DRUG RESISTANCE

Of the 66 cultures set, 42 cultures were positive. Drug resistance patterns observed were as under:

15.3% of our cases were resistant to isoniazid (INH), 10.25% cases to Streptomycin and 7.69% to para-amino Salicylic acid (PAS). Another study showed that the sensitivity status was achieved with a four drug regime for first three months. All patients showing drug resistance of INH alone attained bacteriological quiescence[9].

DRUG TOXICITY

Hepatotoxicity was common in patients receiving INH and Rifampin combined as compared to INH alone. Drug toxicity was more common in patients over the age of 35 years. Stopping of drug for few days and then restoring the treatment without Rifampin is safe line of action[6].

ACETYLATOR STATUS

23.5% of our patients were rapid in activators, 76.2% were slow in activators. There is no direct correlation between development of peripheral neuropathy and acetylator status (Dr. Hajini; Dr. S.N. Dhar, 1975).

DRUG INTERACTIONS

It was observed that antacids and INH should not be given together as antacids significantly decrease levels of INH[7]. Besides some more work has been conducted, which is briefly mentioned as under:

a. Diabetes mellitus and tuberculosis.

b. Association of Pulmonary tuberculosis with chronic bronchitis and corpulmonale.

c. Mycobacterial study of indolent Fibrocavitatory pulmonary lesions.

d. Pulmonary tuberculosis and blood groups

e. Electrocardiographic study in pulmonary tuberculosis.

f. Serum Calcium levels in pulmonary TB.

Our problems with the disease briefly include:

1. Delay in diagnosis
2. Lack of proper public education programme
3. Lack of regular screening programme

4. **Inadequate funding to cope with newer method of diagnosis and treatment schemes.**
5. **Lack of supervised treatment programme to prevent the problem of increasing number of drug resistance cases.**

WHAT POSSIBLE SOLUTIONS ARE PROPOSED

1. **Challenge of rising trends in disease has to be realized and basic research work on this subject be speeded up.**
2. **Teaching at undergraduate and post-graduate levels be augmented.**
3. **Frequent seminars, symposia and work shops be arranged on the disease, Refresher courses be arranged for young doctors.**
4. **Raising public awareness of the disease.**
5. **Adequate funding to deal with problem of modern laboratory back up even at tertiary health care level and augment supervised treatment facilities to prevent drug defaulters and resistance problems.**

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Prof. Wazira Khanam Principal/Dean
Medical College inaugurating CME Programme on Tuberculosis (Nov - 1995).

Scientific Session On T
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Prof. M. Yousuf (Course Director) & Prof Bashir (Chief Guest)
Receiving Prizes from Dr. G.M. Malik (Co-ordinator) &
Ciba team for distribution amoung speakers on Tuberculosis.

Students Page

JK Practitioner 1996; 3:1 60-60

SCENARIO OF TUBERCULOSIS IN SAARC REGION AND W.H.O RECOMMENDED STRATEGY OF TUBERCULOSIS CONTROL.

Tuberculosis is a major public health problem in SAARC Region. 7 Member Countries Bangladesh, Bhutan India, Maldives, Nepal, Pakistan and Sri. Lanka having about 5 million estimated TB patients. 2 million new TB patients coming every year out of which 9,00.000 are sputum positive, responsible for spreading disease in community 700, 000deaths occure in the region due to Tuberculosis every year. SAARC region bearing 25% burden of the world TB problem.

TB CASES IN SAARC COUNTRIES

Country	Estimate number of Sputum Positive TB
Bangladesh	621,000
Bhutan	8,000
India	3,636,000
Maldives	1,000
Nepal	53,000
Pakistan	217,000
Sri Lanka	420,000
Total	4,956,000

Estimated Annual Risk of Infection, incidence and mortality due to Tuberculosis in the SAARC Region

S.No.	Country	Estimated Average	ARI(%) Range	Expected Cases all forms (1000)	Expected deaths all forms (1000	Death\ Rate per (100,000)
1.	Bangladesh	1.50	2.00-1.00	202.8	83.5	68
2.	Bhutan	1.50	2.00-1.00	0.9	0.2	36
3.	India	1.70	2.50-1.00	1641.7	544.0	63
4.	Maldives	1.50	2.00-1.00	0.4	0.05	24
5.	Nepal	1.50	2.00-1.00	33.4	13.8	69
6.	Pakistan	1.00	1.50-0.70	128.2	48.7	38
7.	Sri Lanka	1.50	2.00-1.00	29.4	11.8	67

Assumptions:

1) 50% are smear -positive, In the SAARC region
2) 50 (40-60) smear positive cases per 100000 per each percent of ARI
3) 1.22 Smear-negative and extra-pulmonary case for each smear-positive case
4) Cure rate of the SAARC region is 60%.
5) Case fatality among cases reported but not cured: Smear positive; 40%; Others: 30%
6) Case fatality among cases not trearted; Smear positive; 55% Others; 43%
7) Additional HIV-related cases are taken into account

Source:- Demographic data for Health Situation Assessment and Projections. WHO. Geneva. 1993.

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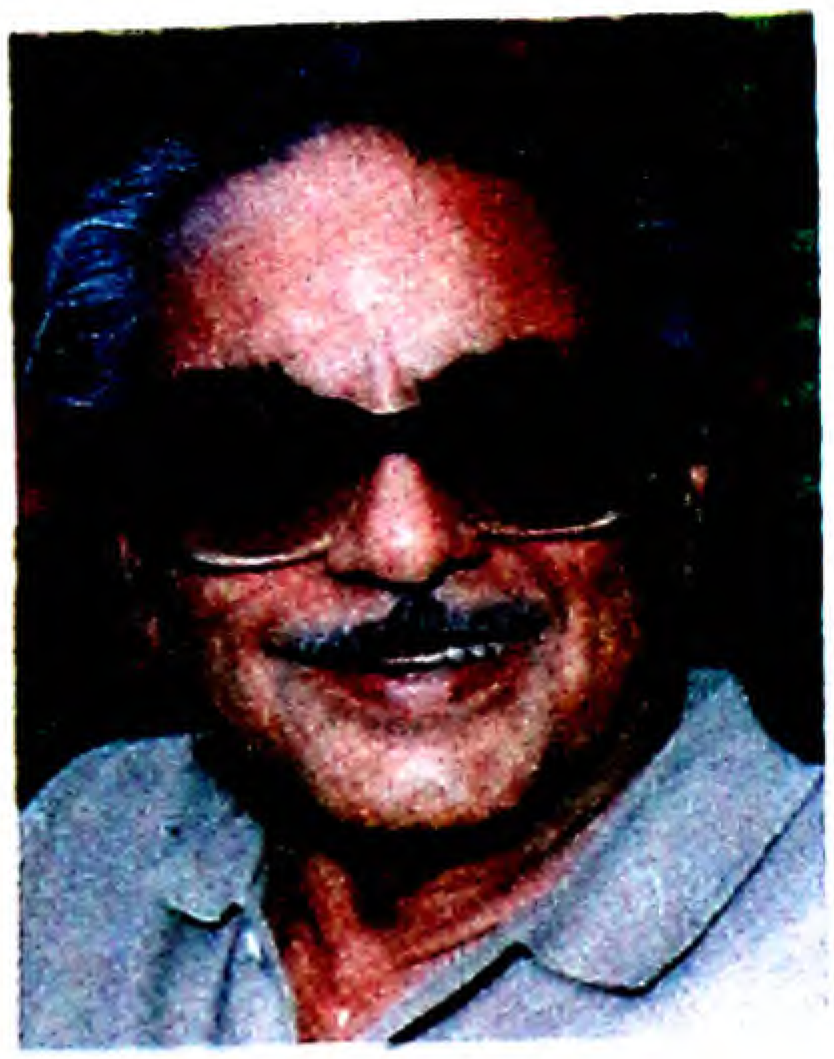
MEET THE PROFESSOR

JK Practitioner 1996; 3:1 61-62

Dr. Syed Naseer Ahmad Shah
Brane, Nishat, Srinagar.



Birth Place:-
Born in 1930 at Boniyar, Baramulla, Kashmir.

Education:-
Schooling in Biscoe School and got his M.B.B.S. from King Edward's College in 1950. Worked in England and did his M.R.C.P from U.K in 1956.
Granted F.R.C.P. for professional excellence in 1978 and F.A.M.S. in 1980.

Appointments:-
Started his career as Consultant Physician in S.M.H.S. Hospital in 1957 Associate Professor Medicine in 1961. He was appointed as Director-Professor and Head of Postgraduate Department of Medicine and **Principal/ Dean in 1969** and left on voluntary premature retirement in November1981.

Additional Posts:-
He has been visiting Professer to many Universities and also to Japan and represented India in many International Conferences. He has been member of the Medical Council of India. Member, Inspection Board, U.G.C, Member, Selection Commitees for U.P.S.C., University of Aligarh and Banaras and on Scientific Advisery Board of Indian Council of Medical Research, member Postgraduate Committee Government of India and as Postgraduate Examiner to the AIIMS, University of Delhi, Jaipur, Punjab, Patna, Kanpur, Meerat, Bombay and other Universities.

Publications/Research:-
He has more than fifty publications to his credit, both in national and international journals and has conducted number of research projects under I.C.M.R/ other agencies. He has been Rockfeller fellow for clinical epidemiology and has contributed chapters in the Text Books of Medicine.

Achievements:-
During his tenure more than 70 postgraduates passed M.D in Medicine and his breaker vision for manpower development is obvious from the fact that all the departments in Srinagar/Jammu Medical Colleges/Institute of Medical Scinces and other Institutions are amnned by his students. He is Member of P.G.I Chandigarh and AIIMS Delhi governing bodies and in view of his dedicated and professional excellence he was awarded **Padmishri in 1983.** As a man and a human being prof. Shah has ver few equals in our medical society. He is selfless, devoted to his profession and the cause of poor and downtrodden.
Prof. Naseer is phrahaps the last representative of that band of astute clinicians who had unshakable faith in clinical methods for diagnosing the disease. Sophisticated gadgets have a secondary place in their arsenal; which is not the case now. He is at present a leading consulting physician with social and professional involvoment in many associations and scientific bodies.

(Written up and complied by Prof. M.A. Akhtar HOD Medicine SKIMS Sgr.)

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From the Chief Editor:

It was a great opportunity for me to meet this eminent Medical & Social personality who has played a pivotal role in the establishment & expansion of major Medical Institutions, hospitals, Health care services & Technical Manpower in J&K. Since hundreds of his fans would like to listen & know about him, I would like to briefly convey his message to them by compiling his views, expressed during a brief meeting with him in connection with this column (on eve of new year issue, Reserved in his honour).

1. Sir, What about your present engagements?
Busy at my clinic mainly, read books & Journals,involved in selection and Advisory committes of SKIMS, PGI etc.

2. What about JK-Practitioner?
I keenly read all issues till now. I am proud of my students and hope it will come up to international standard. Keep it up. My good wishes and help always with your team.

3. A word about Present students & Teachers?
During my time as Principal, selection to M.B.B.S was mainly on merit, but now in whole country it is less on merit, so it would definitely effect on quality of new doctors coming from various colleges. We are seeing less dedicated teachers now than before.

4. A word on T.B. control, as President of TB Associtionn?
We have failed to control rise of TB cases because of lack of drugs availible at periphery, inadequate treatment, lack of proper management at hospitals & now facing resistant cases also.

5. What about AIDS?
I am worried we may face this problem worst than TB in future. Our doctors should work hard to prevent its entry here by making people & Medical personal aware about it from today.

6. During your I decade of Principalship how many court cases you faced?
I faced only one case of a writ petition in connection with MBBS ADIMISSION, but it was rejected by Honb,l justice in one sitting after listening to me.

7. What about patient care today?
Doctors should not forget clinical bedside medicine, do surgery in time where indicated, avoid commercialization of profession & be contented .

8. What about costs of Medical treatment now?
In Metropolitan cities like Delhi, Bombay the bill for routine tests go in thousands & for advanced surgery in lacs. But in Kashmir it is still cheapest.

9. What about doctors abroad?
My students & colleagues are doing good job abroad. I wish they return & sereve here like my colleagues & me.

They will feel guilty at old age like my friends who stayed back in UK. They can a contribute & serve a lot to people here especially in advance tecnology and surgery.

I am greatly thankful to prof. Shah for this brief & relevant talk. God has been kind enough to keep him in good health, alert, with sharp memory, graceful & charming personality still, with same dress (used as our principal), Smile on his face, Proud of his colleagues contributions & ability especially Late Dr. A.M Jan, Dr. G.Q. Allaqband and many others. He has felt deaths of Dr. A.A. Beg and considers it great loss to our community and people,

OBITUARY

The Editorial Board, Medical College faculty & teachers Association mourn the death of some colleagues who passed away during 1995.

1) DR I.P. KOHLI.
A noted ENT specialist and former Prof. & HOD ENT. Deptt Medical College Jammu died on 24 Nov. 95 after a short illness. He way 58 and hailed from Kathua.

He did his M.S in ENT at A.I.I.M.S, & joined Medical College Srinagar as Lecturer ENT & afterwards became HOD for long time and then retired as HOD ENT at Jammu Medical College. His death has been widely mourned in Medical & Social circles at Srinagar and Jammu.

2) DR. MAQBOOL M.D:-
A young doctor from Ganderbal, was working in SKIMS after Post-graduation (M.D) in Medicine at Medical College Sgr. He passed away after short illness at SKIMS.

3) DR. MEHMOOD:-
A senior doctor working at Health services Deptt. at Islamabad, was killed by unknown persons.

4) The Chief Editor and Editorial staff also conveys condolences to the bereaved family of Haji A. Rahim Khuroo (Father of Prof. M.S. Khuroo) Sopore and family of Haji M. Yousf Mir (Father of Dr. Mir Nazir) Hyderpora Sgr. who died in Dec. 1995.

From the Archives

JK Practitioner 1996; 3:1 63-65

"FOUNDATION OF FIRST MEDICAL COLLEGE IN KASHMIR" (1959)

***Parveen Chesti (1st Batch Student)**

Till the year 1959 the State of Jammu and Kashmir did not have a Medical College and a handful of eligible students were nominated year after year for admission to different colleges outside the State for medical studies against a limited number of seats that were reserved for the State nominees. With the expansion of health care activities and consequent demand for more qualified doctors, establishment of a medical college in the State became an unavoidable necessity. But the idea of starting one looked like a dream for many years because of lack of resources, infra-structure and availability of teaching staff from within the State. However, to our pleasant surprise it was learnt that a gentleman - Col S Gargey by name - had joined as Advisor in early 1959 to plan a medical college in Srinagar. One could hardly believe that the dream was coming true when in July 1959 a notification was issued by the Advisor inviting applications for admission to the College which had been scheduled to be commissioned the same year. I and my class fellows who had just completed the Intermediate (now 10+2) examination and were eligible for admission to MBBS course had been in touch with some medical colleges outside the State for our admission, but we did respond to the Advisor's notification - of course with our fingers crossed. We had to put in some effort in locating the office of the Advisor, which he ultimately found in a small house in Shahid Gunj, which was both his office and residence. We saw Col. Gargey in his modest office handling the blue print of the college. While welcoming us he presonally handed over to each of us an admission form. We were called for interview in his office in the first week of August and the select list of students was issued within a fortnight or so after the interview. The classes started by the end of August. Sixty students were taken from Jammu and Kashmir and seven from other States. At the interview we had the previlege of getting introduced to the Principal designate, Dr. GVS Murthi, and the Head of the Department of Physiology, Dr. BS Kahali, who had just joined. In record time some space was improvised in the old hospital at Hazuri Bagh (now Lal Ded Hospital) to provide for our class rooms, laboratories, museum and dissection hall. Girl students from our stations were provided residence within its premises and the boys were lodged in a neighbouring private hotel - Everest Hotel, Goni Khhan. Other staff members who had joined by that time were:

Anatomy KK Bhat, Prof. & Head, D Kumar, Assist. Prof., Dr. BM Bhan, Lect., Haroon Rashid, & ML Kaul, Demonstrator, ***Physiology*** BR Tandon, Assist. Prof., Anand, Demonst. ***Bio-chemistry*** SK Dasgupta.

The laboratories were equiped on modern lines.

Col. Gergay left the job and State in November, 1959 after fully accomplishing his assignment. We managed to carry on in the improvised accommodation of the Old Hospital building while construction of the College building on the complex of Sri Maharaja Hari Singh Hospital (SMHS Hospital) at Karan Nagar was in progress. The first teaching block was inaugurated in 1961 in which we started courses for the second professional. At the same time the existing

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* HOD Pathology
Medical College Srinagar
Kashmir - 190 010

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SMHS Hospital was strengthened with the addition of two more wings taking the bed strength to 700 to facilitate clinical teaching. The pioneer teaching staff that had been added for teaching of para-clinical and clinical subjects by 1961 was as under:

Pathology BN Dutta, Assoc. Prof. & Head, DN Thussu, & HK Kaul, Demonstrator, **Pharmacology** (Mrs) Sannan, Assoc. Prof. AH Fazili, Demonstrator, **Forensic medicine** TN Malla , TR Amla, Demonst., **SPM** Anantha Rao, Prof. & Head, Avtar Singh Sethi, Assist. Prof., **Paedriatics** Thaman, Prof. & Head, **ENT** Harbajan Singh, Assoc. Prof., IP Kohli, Assist. Prof.,**Opthalmology** Harbajan Singh,Assoc. Prof., Subhash Gupta, Assist. Prof., **Medicine** S Kaul, Prof. & Head, SP verma, Prof., Ali Mohammad Jan, SN Ahmad Shah, Assoc. Prof., Khawja Ahmad, SL Varma, Assist. Prof., **Gynaecology & Obstetrics** (Ms.) S Malhotra, Assoc. Prof., (Ms.) S Arora, Assist. Prof., CS Khanna, Prof. & Head, **Surgery** Parmanik, Prof., Ghulam Rasul, Girdahari Lal Kaul, Assoc. Professors, Sannan, BM Bhan, Assistant Professor.

It may be mentioned that Dr. Ali Mohammad Jan, Dr. SN Ahmad Shah, Dr. Ghulam Rasul, Dr. Girdhari Lal Kaul, Dr. Garbajan Singh and Dr. (Miss) S Melhotra, who were working as Senior Specialists in SMHS Hospital when the College came into being, were designated as Associate Professors of the College in their respective specialities. Later on all of them were elevated as Professors. Dr. SN Ahmad Shah worked as Principal also for over a decade from 1970. The teaching staff that was recruited from outside the State was given higher salary and provided free residential accommodation in the city and other attractive perks in view of their high calibre. Their services were taken on contract basis so that they could be replaced by local staff on becoming eligible to occupy higher positions under the Medical Council of India rules.

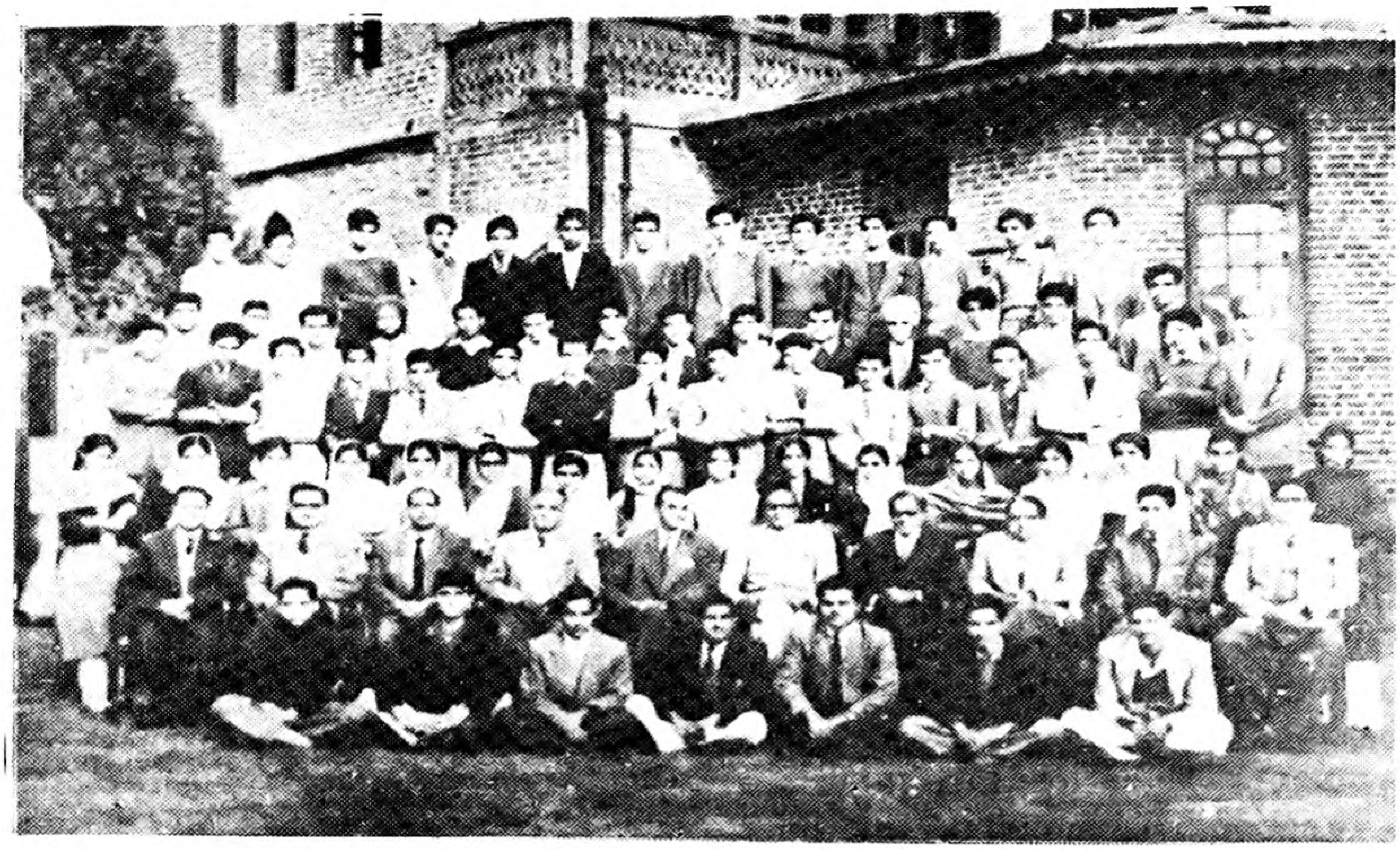
The College had thus the unique previlege of having such personalities of eminence and rich experience on its faculty to lay its strong foundation and set high academic excellence to the extent that from its very infancy the College came to be known as one of the most prestigeous medical Institutions of India.

Another feature of the College from the very beginning was that it provided facilities for extra-curricular activities. Debates, plays, musical concerts, cultural activities, sports and games were encouraged and cultural and sports functions were held every year of a large scale before distinguished gatherings. The first sports day was held in the Polo ground in July, 1960.

The duration of MBBS course in our time was five years followed by 6 months internship. The University examination of final MBBS was held in June-July, 1964 and the internship completed in January, 1965 strictly according to the schedule that had been drawn at the time of our admission. All of us joined various branches of medicine of our choice immediately after graduation. Presently several graduates of the first batch occupy positions as Heads of different departments in the two Medical Colleges in the State and outside the State. One of them is working as Principal of Jammu Medical College. Some of them hold top positions in their respective fields in advanced countries like UK and USA. The graduates of other batches who followed the first batch also enjoy high reputation for their outstanding talent and contribution to different branches of medical sciences, both in India and abroad. We would have hardly stretched our imagination, while dreaming a Medical College in Srinagar a few decades back, to the extent of seeing its products reaching such heights.

The college celebrated its 1st Silverver Jublee in 1984 under the chairmanship of Prof. S.Z.Ahmed Principal Medical College Sgr. Prof Ashai was the chief organisor for all the delibrations during the whole week of silver jublee programme.

Ist batch of Staff of students (1958) Medical College Premises (Courtesy Dr. Chesti)



Sitting (L-R) Ashok, M. Shafi, Bal Krishen, Farooq A, M. Askari, Prem Kamal, Kustoori Lal. (Principal Jammu)
Staff (On Chairs) T. R. Anand, B. M. Bhan, R. C. Tandon, B. S. Kohli (HOD Physiology), Col. K. S. Garg (Advisor), GVS Moorti (Principal), K.K. Bhat (Head Anatomy), D. Kumar, Haroon Rashid, M. L. Koul.

1st Cultural Show in Medical College (Students of 1959 are Parents in 1995)

IN MEMORIUM

JK Practitioner 1996; 3:1 66-66

Dr. Farooq Ashai (1938-1993)

Professor Farooq Ashai was born on 13th April, 1938 in Ashai Mohalla Fateh Kadal, Srinagar. He had his early education in M.P High School and completed his MBBS in Amritsar Medical College in the year 1961. He did his postgraduation in Orthopaedics from Patna in the year 1965 to become the first Kashmiri to be an Orthopaedic surgeon and joined as Assistant Professor to finally became Professor Orthopaedic department 1972. He published a number of research papers in Orthopedics and also authored a book on bone and joint injuries. He ensured upgradation of orthopedic section under general surgery to a full fledged independant 250 bedded Bone and Joint Hospital at Barzulla which was his personal achievement. The entire Hospital was conceived engineered and constructed by Professor Ashai under his personal supervision. He also creared an artificial limb centre in the premesis of the bone and joint Hospital. He was an excellent administrator and served as a Medical Superintendent of Bone and Joint Hospital and Director of the A.L.Centre. Professor Ashai organized a number of scientific conferences, and published a number of scientific papers in various journals. He had great love for music and peotry, played violin, harmonium and tabla like a qualified artist. He was an excellent teacher and a gifted clinician. He always appeared not to freely mix with people and even his colleagues though was very intimate with a few selected ones. A great freedom fighter, was martyred in cold blood on the fateful day of 18th February, 1993. May his sole rest in paece Amin.

(Contributed by Prof. G.H. Hajini HOD Dermatology GMC, Srinagar)

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I have mustered the courage to write for a person whose command on English and method of writing have far exceeded that of a student of this language. I dear to do so only in his absence.

Such a wonderful teacher, lever of precision, fine art, clean habits and high morale made him an institution in his own right. His short text book on Bone and Joint & Injuries testifies to the above facts. Being trained at Amritsar AS AN Undergraduate student, he had seen the services of professor karam-Singh for the people of Punjab and their reciprocal love and gratitude inspired him into orthopedics with a promise that he would do something for his people in Kashmir. He did this but with little extra;gave his life.

Brief encounter with Prof. Duraswami; as a House Surgeon and prof. Tuli then as his Registrar at Delhi, he eventually get trained in Patna by none other Prof. B. Mukhapadaya. Having already grasped the basics of spinal surgery especially spinal T.B., he went to Milwaukee U.S.A. and learnt from Dr. W. Blount further tricks of the trade. His contributions in Orthopedics as a pioneer in the State ar eun-parrelled and have significance as milestones. As his House Surgeon and Registrar from 1968 to 1971, I have seen his interior surgical procedures for spine and we have added only little to them. he had undertaken to develop spinal surgery after commissioning the Bone and Joint Surgery Hospital, but untimely death did not permit him to proceed further. On the fageful day while washing my master's feet, I vowed to bear the responsibility he had undertaken namely to look after his Institution not as a place but with its traditions and rising standards; things he loved most and continue his unfinished work.

To replace him is not possible but adoring what he loved and left behind would be what he himself would approve of. May Allah give give him abode in "FIRDOUS"

(Contributed by Dr. Mir Gh. Rashool FRCS Prof. of Bone & Joint Hospital Barzalla)

IN MEMORIUM

JK Practitioner 1996; 3:1 67-68

Late Dr. Abdul Ahad Beig (1943-1995

Dr. Abdul Ahad Beig was born in a modest Kashmiri family on July 17, 1943 at Bandipora, District Baramulla, Kashmir. He was a brilliant student from his early student days. He passed his M.B.S. from Government Medical College, Srinagar and subsequently obtained his M.D. degree from the Department of Psychiatry, P.G.I.M.R., Chandigarh in 1976. he joined the Department of Psychiatry, Government Medical College , Srinagar as Registrar in September 1976 and was appointed as lecturer in Psychiatry in July 1977. He was promoted as Assistant Professor in March 1981 and Associate Professor in March 1980. In March 1995, he joined medical College, Jammu as Professor and HOD Psychiatry and in the same year was transferred back to Department of Psychiatry, Medical College, Srinagar as Prof. and Head, also the Medical Suprintendent of the Hospital Psychiatric Diseases, Srinagar. Unfortunately on November 29, 1995 when he was about to leave for Jammu alongwith his family on a brief holiday, he was struck by some febrile illness and passed away on December 5, 1995 at the prime of his distinguished career. He has left behind his young ailing wife and three teenage children which include two daughters and a son.

In many ways as the forerunner, Dr. Beig created a name for himself in the field of psychiatry in the state of Jammu and kashmir. The pioneer not only contributed to training the present generation of psychiatrists of the state but also played the pivotal role in freeing, to a large extent the treatment of psychiatric disorders from the dominance of witch craft and faith healers. Through his dedicated clinical practice, he succeeded in increasing the awareness about psychiatric ailments among the people in general and physicians in particular in the valley. Dr. Beig

was a life fellow of the prestigious Indian psychiatric Society and cherished the dream of enriching the academic and research activities in the field of psychiatry in the endeavors and compile the results of his vast clinical experience and knowledge, a cruel blow has been dealt to future prospects of psychiatry in Kashmir owing to his untimely death.

Throughout his career Dr. Beig wore a charming smile on his face, generously wiping tears from eyes of others and benevolently pulling out dejected, depressed and disorganised persons from dispare and despondency, guiding them to lead an honourable life. The Holy Prophet has affirmer ‘ Do not look upon pious deeds as insignificant, may it be greeting your brother with a cheerful heart’ (Muslim). On another occasion the Holy Prophet (PBUH) observed that a man who bears a sweet temper, tender heart and is soft spoken will enter into paradise and for him the fire of hell has been made unlawful (Tirmidhi). Sweet temperer, Tender hearted and soft spoken Dr. Beig remained throughout his life dedicated to his family members, friends, Colleagues, Students and patients alike. He led a simple life and was a firm believer in God. His charismatic personality was a real reflection of the magnificent features of Islamic life of civility and good manners, dignity and courtesy, prudence and discretion, organization and discipline, keen aesthetic sense, magnanimity and nobility of temperament, sympathy and consideration mildness and pleasant speech, hospitality and humility, selflessness and sacrifice, Lustlessness and sincerity, fortitude and perseverance, sense of responsibility, bold initiative and above all reliance on fear of God. It was because of such qualities that people form different classes and communities after talking to him just once would get irresistibly drawn to Dr. Beig as their friend philospher and guide.

I had a close association with Dr. Being as his student, colleague and as an associate. I admired his excellent and meticulous way of teaching, his noble way of living and his personal affection for me. May his noble soul rest in peace-Ameem.

(From Dr. Mushtaq A. Marghoob, consultant psychiatrist, G. M. C., Srinagar, Kashmir).

In memory of Dr. A.A. Beigh (short poem)

Is it demise or disaster,
Is it death or departure;
I see it death or demise never,
but an annoyment for ever,
Will you come back for a while
To see you notforgotten but alive.
Your kids still just buds
Your wife now without life
They wait for you, to come to,
Knowing that it never happens so
To forget you, tell them what to do.
Your family, your relatives,
Your colleagues, your friends, your patients
and the hearts of the valley,
all are very sorry for you.

From Dr. Hamidullah Shah acting HOD Psychiatry.

It is with deep sense of sorrow and grief, I have to say that we have lost Dr. Beg Sahab at a time when we needed him most. Dr. Beg Sahab was a man of great depth and wisdom an embodiment of nobility, with a deep insight into pain and suffering of his fellowmen. He was a Messiah as a Psychiatrist with a miroconious human and healing touch. In him I had found a great friend whom I have last though I would always remember his friendship with great pride and honour. May Allah Bless the departed soule (Amin)

Prof. Mohammad Yousuf (Deptt. of Medicine GMC)

A soft spoken, ever smiling, young handsome man, an excellent neighbour. Dr. Beg, was a great friend of mine. His loss to the society, in the present situation, is irrepairiable.

Prof. M. Amin Akthar (HOD Medicine SKIMS)

Similar views & remarks were expressed by number of his colleagues friends. namely Prof. Hajini, Prof. A.R. Khan, Prof. Ahad, Prof. Bashir, Prof. Nafi. Prof. Bilquis, Prof. M. Ashraf, Dr. Zaffar Mehdi, Dr. G.Q. Khan, Dr. H.U. Malik, Dr. Ashaq Hussian, Dr. Kawoosa and others like Medical College Teachers Association Srinagar as well as Jammu.

It is really a good gesture that January, 1996 issue of the J.K. PRACTITIONER has been dedicated to the memory of late Prof. A.A. Beig, Head of the Department of PSYCHIATRY of the Government Medical College, Srinagar at the instance of the Chief Editor, Dr. G.M. Malik. Dr. Beigh was respected by the teaching faculty of the college and the students. The untimely passing away of Dr. Beig will be remembered for all times to come as a colleague, a teacher, an eminent clinician and a social worker. The community needed his services to expand the speciality as well as help ailing patients which are on increase due to the prevailing circumstances.

May God grant peace to the departed soul and sustenance to the bereaved family to bear the loss.

Dr. Wazira Khanam, M.S
Principal/Dean GMC Sgr.

From the Editor:

I lost a close friend, clinician, a great writer and a social worker. May God give peace to his soul & courage to his family to bear this irreparable loss.

JK Practitioner 1996; 3:1 69-70

OVERSEAS DIARY:

History and Training of Foreign Medical Graduates in U.S.A

Faroque A. Khan, MB, FRCP(C), Chairman Medicine Deptt. Nasu County (USA)

Approximately 650,000 physicians reside in the United States. Of these, 144,399 are IMGs (22%), of whom approximately 85% are practicing in predominantly underserved areas and 15% in academic medicine. Many IMGs have achieved considerable success in science and medicine (andare, in fact, Nobel laureates, academic leaders, physician scientists, clinician teachers, and community leaders). These IMGs tend to concentrate in large cosmopolitan states. In 1992, New York had 26,059 IMGs, California had 15,666, Florida had 10,996, New Jersey had 9,757, and illinois had 9,741. The states with the fewest IMGs as of 1992 were Wyoming with 43, Idaho with 44, Alaska with 50, Montana with 72, and Vermont with 109. in New York, IMGs represent approximately 50% al all licensed practicing physicians. As a group, IMGs are heterogeneous; however, IMGs are often categorized into one group, which results in a flawed analysis and a resulting pattern of unfair discrimination (1). Patterns of entry of IMGs in the United States are often determined by the perceived needs for physicians in the United States and the sociopolitical situation in other countries. For example, during the Vietnam War a large influx of IMGs was caused by a perceived physician shortage in the United States. Similarly, after the collapse of communism and the breakup of the USSR, many IMGs came to the United States from Eastern Europe. The major events marking the entry of IMGs to the United States are shown in the table below.

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HISTORY OF INTERNATIONAL MEDICAL GRADUATE ARRIVALS IN THE USA

1933-1948, European IMGs immigrate as refugees in relatively small numbers
1948, Exchange Visitor Program allows IMGs to train in the United States, and many of these physicians stay.
1956, The American Medical Association and other organization begin an IMG-certification system.
1965, Visas are obtained more easily for physicians working in some specialties, which attracts IMGs from developing nations.
1971, IMGs obtain job clearances for permanent residency status more quickly.
1976, Congress introduces immigration barriers against IMGs.
1980, A federal study recommends IMG limits
1985, Federal legislation is proposed to curtail graduate medical education funding for IMGs, but it fails.
1990s, Steep increase in incoming IMGs is attributed to the breakup of the Soviet Union, changes in licensing examinations, and new immigration law

The specially selection of IMGs also follows a predicable pattern. With most working in primary care specialties. For example, in 1992, of the 144,399 IMGs, 27,941 (19%) were practicing in internal medicine, 14,080 (10%) were practicing in pediatrics, and 9,847 (7%) were practicing in psycychiary. Thus, almost one half of all IMGs work in primary care specialties.

International Medical Graduates as Trainees

In the past decade, U.S. undergraduate medical school enrollment has remained relatively stable. However, the number of graduate medical training positions has increased steadily from 73,783 in 1981 to 86,566 in 1991, an increase of 21% (3). The number of residents has increased correspondingly from 69,738to 86,217, reflecting a rate of growth of 24%. For internal medicine, the percentage of first-year residency positions filled by U.S. medical graduates has decreased steadily from 86% in 1976 to 63% in 1992 (4). New York State constitutes 7% of the U.S. population, graduates approximately 10% of U.S medical students, and trains approximately 15% of the medical residents. In fact, New York postgraduate year-1 position equal 165% of the total graduates from the 12 New York allopathic and 1 osteopathic medical schools. Generalist residency positions are filled New York by more IMBs than U.S graduates. In 1993, of the approximately 2,000 first-year positions that were filled in New York in medicine, pediatrics, and family medicine, approximately 780 (39%) were filled by U.S. medical graduates, and the rest , 1,220 (16%), were filled by IMGs, both through the National Resident Matching Program and outside it. Nationally, 36% of all internal medicine training positions are filled by IMGs.

IMGs are a heterogeneous group of physicians from approximately 1,400 medical schools. They represent a diversity of cultures, backgrounds, ethnicity, languages, and educational systems; their diverse outlooks have evolved in response to critical regional issues. The education and certification of physicians within a countries country involve a complex interaction among political, educational, and health care delivery systems. That interaction can very widely among countries within a geographic region (as witnessed by the contrast between the health care delivery and medical education systems in the united States and Canada). A detailed review of the current status of medical education around the world, as individual nations train physicians for the 21st century, is found in the recently published International Handbook of Medical Education (5). To develop uniform systems to evaluate IMGs who seek further education in the United States, the Educational Council for International Medical Graduates was established. The first screening entrance examination was given in march 1958 in 17 centers, and 298 candidates were tested.

From 1958 to 1994, the Educational Council for International Medical Graduates examined more than 500,000 candidates and issued 241,556 certificates to candidates who successfully passed the examination and fulfilled other requirements of English proficiency and local registration in their country of origin. Many of these IMGs have settled in the United States, and approximately one quarter of the more than 600,000 physicians here received training overseas. Of the more than 125,000 IMGs, about 15% are in academic medicine and the rest are practicing in urban and largely underserved areas. These IMGs comprise 26% of practicing internist, 36% of internal medicine residents, and 21% of the membership of the American College of physicians. Eighteen IMGs have been Nobel laureates, whereas other have been authors, administrators, and program directors, and they comprise 50% the physicians working at the National Institutes of Health. In the 1990's, most IMGs who participated in the Educational Council for international Medical Graduates examination originated from India, Pakistan, and the Philippines. For example, in 1993, that organization issued 10,815 certificates; of these, 2,721 (25%) were from India, 1,208 (11%) Pakistan , and 854 (8%) from the Philippines. Thus, these three countries provided 44% of all IMGs for training. These trainees tend to be concentrated in New York (1,176), California (830), New Jersey (471), Illinois (416), and Florida (317).

My Experience

I graduated from medical school in Srinagar, Kashmir, and was actively recruited by a community hospital in Barberton, Ohio. The hospital paid my airfare from Srinagar to Barberton. My wife and I were met at the Akron, Ohio, airport by an attending physician. We were driven to a furnished apartment, we had a supply of food and $100 as emergency money.

CORRESPONDENCE AND COMMUNICATION COLUMN:

Dear Editor,

Thank you for sending me the recent editions of the JK Practitioner. While the journal has been improved, there are still considerable areas which need to be updated - quality of paper, re-production of pictures, and editing are some are as that come to mind.

I am enclosing a complimentary copy of the book International Medical Graduates in U.S Hospital: A Guide for program Directors and applicants for your use and reproduction. Hope you find it useful.

Dr. F.A. Khan,FRCP FCCP
Chairman Medicine, Nasu County Hospital USA

Dear Editor

This letter is intended to introduce to you "The Indian Journal of Chest Diseases and Allied Sciences" Which is now in the 37th year of publication. The Journal is published under the joint auspices of the National college of c\Chest Physicians (India) and the Vallabhbhai patel Chest Institute, University of Delhi. It has a wide national and international circulation. We shall be grateful if you could consider the exchange of your esteemed Journal, "JK practitioner" with ours. Looking forward to a favourabe response, and with kind regards,

Prof. A.S Paintal
Editor-in-Chief
Indian Journal of Chest Diseases &
Allied Sciences, Vallabhhai patel
Chest Institute, Delhi.

Dear Editor,

In response to your request, I am submitting herewith an article entitled, " Current Concepts in the management of Gallstones disease". I hope you find this useful and publishable for your excellent journal' JK Practitioner".

Prof. Rakesh Tandon
Professor & Head Deptt. of
Gastroenterology, AIIMS Delhi

Dear Dr. Malik,

I am very glad that the issues of practitioner (international) are coming out very regularly.

The credit goes to your dynamism & energy. Keep it up.

Prof. U. Kaul
Prof. of Cardiology
All India Institute of Medical Sciences Delhi

Dear Dr. Malik,

I am afraid you have misunderstood my previous letter. I cannot commit myself to writing a review article at the moment as I have so many other editorial commitments, and certainly not by the time you mention.

In the meantime, I enclose a copy of a recent article from this institute which I hope you will find interesting. If would of course need to apply to both the British medical Journal and the authors here for copyright permission.

Professor Roger williams, CBE
Director, Institute of Liver Studies,
Kings College London, University of London.

Dear Chief Editor,

Thank you for your kind letter. Enclosed is a small selection of articles from my published work that may be of interest to you.

I admire your efforts and wish you the very best in all your laudable endeavors on behalf of the community.

Prof. P.K. Shah
M.D. FACC
Director Division of Cardiology and
Atherosclerosis Research Center
Prof. MedicineLos Angles, (USA)

Dear Editor,

It was great to know you, but the time was so short. You are one of the person in Kashmir whom I saw with a scientific & experimentation mind, besides giving Kashmir the Ist ever Medical Journal. I hope your plans succeed & the next generation of doctors be proud of yourself.

Dec. 95 *Dr. RWOOFA RESHI*
Scharm burg illumors, Chicago (U.S.A)

Dear Chief Editor,

I hope you must have received the previous articles submitted by me to your office for publication in your esteemed Journal. I am realy proud that you are doing so much work in such a difficult times and without much local support. I think we should all work very hard to keep this journal going on and make it one of the reputable Journals.

31.10.95 *Dr. N.A. Bhat*
M.S. FRCS.
10-Torrant walk
Walsgrave Coventy (U.K.)

Dear Dr. Malik,

Thank you very much for your letter of 28th of September 95 I look forward to seeing the next issue of your Journal. I will be asking a medical student here to write up a diary of a student in this country which may be suitable for publication in your journal.. I can also arrange to send you some from Russia, America and Europe and I think it may be a good idea to have a diary from these various places either in each issue or evey other issue. This would give your readers some idea as to how medical education is progressing in other countries and it may be of interest to the young readers who are currently undergoing medical training in kashmir, i notice that you do not have a correspondence section in your Journal. I think it may be a good idea to have a brief correspondence section about the articles published or about any other issue that your readers might find of interest. If there is any thing I can do in the future please do not hesitate to let me know.

Dr. M.A. Mir,
Senior Lecturer & Consultant Physician,
Department of Medicine, University
Hospital of Wales, health Park Cardiff
CF4 4XW, U.K.

Dear Editor,

I whole heartedly congratulate you for this well deserved and over due publication of this Journal especially from a community of intelligentia of this country. The get up is very good.
Wishing you all the best for new year.

Dr. Lt.Col. Kotwal M.D. FACG
Medical Avisor cum Chief Consultant
Govt. of Sikkim.

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I Dr. G.M. Malik hereby declare that the particulars given above, are true to the best of my knowledge and belief.

Sd/-
Dr. G.M. Malik

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Editorials:

-Radio-Iodine and Thyrotoxicosis. 73 AH Zargar.
-Aids Dementia. 74 PA Shah, A Nafee.
-Current Postgraduate Curriculum- Need for Improvement. 75 A Rouf.
Guest Article:
-Percutaneous Cardiopulmonary Support in high risk interventions. 77 FA Shawl.
Clinical Review:
-Diet and Colorectal Cancer. 82 NA Wani, OJ Shah, T Fazili.
-Approach to Child Health. 85 GM Dar.
New Horizons in Non-Invasive Surgery :
-Extra Corporal Shock Wave Lithotripsy(ESWL). 88 MA Khan.
Origional Articles:
-Thyrotoxicosis, Management with Radio active Iodine (A Follow up study). 90 S Mushtaq, SH Khan.
-Effect of Fluoride ions on the thyroid glands of Guinea Pigs. 94 Mahmood, GH Bhat.
Case Reports:
-Massive Splenomegaly, an Unusual Presentation of Echinococosis. 97 SA Aziz, M Ahmed, MA Amin, GM Bhat, A Lone, G Nabi, H Bano.
-Adenoma Upper Lip (Papillary -Type). 99 B Ahmed, M Shafi, N Rashid.
-Body Favus. 100 Q Masood, S Manzoor.
-Mexiletine Toxicity. 101 SB Bansal, HS Rissam, FA Bhat, J Ashraf.
-Tuberculous Parotitis. 102 NA Bhat.
Special Article:
-Organ Transplantation. 104 MA Albar.
Practitioners Column:
-Allergy and Chronic Allergic Rhinitis. 109 MM Maqbool.
-AIDS in Children (Part III). 111 GH Hajini.
Students Page:
-Cosmetics in Practice. 113 S Manzoor, S Parveen.
Clinical Images: 116 AH Zargar.
Hospitals Today:
-Performance Appraisal A Subjective Phenomenon in Hospitals. 117 MA Wani.
Letter to Editor:
-Heridditary Orotic-Aciduria can be caught. 119 ST Hussain.
-Pulse Polio Campign in valley. 120 MA Shawl.
Meet the Professor:
-Prof. M. Maqbool (ENT). 121
-Prof. Upendra Kaul (Cardiology). 122
In Memorium:
-Late Prof. A. A. Guroo. 123 A Ahad, Ashaq Hussain.
Overseas diary:
-Life as a Medical Student in th U.K. 124 Monica Mathur.
-Issues faced by IMGS and Programme Directors in USA. 126 FA Khan.

Communications to editor:

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Main Topics in Forthcoming Issues (July-Sept. 1996)

-> **Post - MI Mechanical Complications.**
-> **Chronic Hepatitis.**
-> **Biochemical Assessment of Hepatic Billiary Disorders.**
-> **MR Imaging in Proptosis.**
-> **Liver Transplantation.**
-> **Cancer Prevention (Part I).**
-> **Diabetic Retinopathy.**
-> **AIDS (Part IV).**
-> **Ultrasound Images of Stomach**
-> **Doctor Patients Relationship.**

Editorials:

"Radio-Iodine and Treatment of Thyrotoxicosis"

JK Practitioner 1996; 3:2 73-74

Radio-iodine (RI) is the simplest and the most economical means of treating thyrotoxicosis.Two types of isotopes have been used for the treatment of thyrotoxicosis I^{131} and I^{125}. Theoretically I^{125} would be more effective in treating the thyrotoxic state without causing subsequent hypothyroidism. However long term follow up of patients has revealed a frequency of hypothyroidism equal with both the isotopes [1,2]. Controversy still exists regarding the age of patients, dose of radiation given and etiology of thyrotoxicosis. RI is considered treatment of choice in patients with toxic multinodular goiter or toxic solitary nodule as these states usually occur in patients of more than 40 years of age. Because Grave's thyrotoxicosis occurs at young age, more in females and has an immunologic etiology, antithyroid drugs and surgery are still considered a treatment of choice in these patients.

The most consistent consequence of RI treatment for thyrotoxicosis is the subsequent development of hypothyroidism. The frequency is more in the initial two years after RI treatment and subsequently increases by 3 percent per year [3]. Frequency of hypothyrordism is more in elderly patients, those with thyroid antibodies and varies directly with dose of RI given [4]. In the article entitled "Thyrotoxicosis: Management with Radio active Iodine (1-131) follow up study in 174 patients". authors have discussed the role of RI therapy in management of thyrotoxicosis of different etiologies. Authors have given a fixed dose of 10 mcu of 1-131 in the treatment of thyrotoxicosis and found that patients of Graves disease and solitary toxic nodule showed a 100% response. Some comments need to be clarified on the design and outcome of study. Author has measured the size of the gland and nodules manually. Thyroid gland can be staged manually for epidemiological studies, but this staging is not applicable to the small patient population which attend the hospital. It is not possible to measure the size of nodule manually. Authors has divided the patients into three groups viz; Grave's disease, solitary toxic nodule and toxic multinodular goiter without mentioning how one group of patients was separated from the other. A patient may clinically have a single nodule but actually has TMGN. Similarly in an endemic iodine deficient area patient may already have a multinodular goiter but subsequently develop Grave's disease. Grave's disease is immune mediated disease and occurs at young age, is more frequent in females. Most of the authors still recommend antithyroid drugs/or surgery for this patient population. Recurrent thyrotoxicosis after surgery forms another indication for RI therapy. Radiation thyroditis is a significant complicetic of RI treatment and an occasional patient may develop thyroid storm.

Kashmir valley has been documented to be an iodine deficient area. Here almost half of the children have iodine deficiency goiter [5-7]. In an iodine deficient area the incidence of thyrotoxicosis steeply increases after iodine suplementation [8]. Similarly in an iodine deficient are TMNG has been shown to be resistant to RI therapy that could be the reason why patients of TMNG need more dose of RAI.

Keeping in view the varying etiology and controversy about age patients should be taken for RI therapy only after proper selection and these patients also need to be followed for radiation thyroditis and long term hypothyrordism.

Dr. A. H. Zargar MD, DM
Associate Professor and Head
Endocrinology Deptt. SKIMS,
Soura, Srinagar, Kashmir- India.

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"AIDS - Dementia"

JK Practitioner 1996; 3:2 74-75

Human Immuno deficiency virus (HIV), the aetiological agent of AIDS apart from affecting the immune system, also has prediliction for central nervous system. Nervous system involvement in AIDS manifests as dementia, opportunistic nervous system infections, myelopathy and peripheral neuropathy. Annual incidence of HIV dementia is about 7% of U.S patients with AIDS and is the only initial manifestation of AIDS in less than 3% cases. Reardon et al found dementia to be more common among older gay and bisexual men than among intravenous drug abusers, Hispanics, Haitians and women.

It has been speculated that there may be a specific neurovirulant strain of HIV responsible for causing dementia. The V_3 envelope region of HIV is an important determinant of neurotropism. Long terminal report (LTR) region of HIV genome, which regulates viral replication may also be a determinant for neurotopism.

Predictors of HIV-dementia before onset of full blown AIDS are as follows:-

- Anaemia - Low body mass index - Old age - Presence of constitutional symptoms - Presence of HIV-p_{24} antigen in the CSF - Presence of (B_2) - microglobulin (>3.8mg/l) in CSF in absence of any opportunistic infection - Increased levels of prostaglandin E_2, Neopterin and quinolinic acid in cerebrospinal fluid.

The mechanism of neuronal dysfunction in AIDS dementia is probably multifactorial and may be discussed as follows:- Cytopathic effects of the reterovirus by causing activation of macrophages/ microglia, neuronal damage mediated by viral proteins-gp120, nef, tat; other mechanisms responsible for neuronal damage include monokines (TNF-α) mediated neurotoxicity; blood-brain barrier perturbation with consequent exposure of parenchymal cells to infected monocytes and possible interference with "astrocyte-derived neuronal protective factor" by gp120. Moreover, other infectious agents like cytomegalovirus, and JC virus may stimulate transactivation of HIV. TNF-α may also stimulate transactivation of HIV. The neurotoxicity of gp120 is believed to be mediated by NMDA receptors or release of nitric oxide. HIV gene products "Nef" protein (involved in down regulation of viral expression) is expressed in higher quantities in demented patients and "tat" (geneprotein involved in up-regulation of viral replication) has been found to be neurotoxic in tissue culture.

Infection of the brain by HIV results in distinct neuropathological changes termed "HIV encephalitis", "HIV leuko encephalopathy" and diffuse poliodystrophy" which account for 40%. 33% and 50% of HIV dememtia patients respectively. Histopathological examination reveals perivascular infiltration of monocytes, macrophages, and microglia. Levels of TNF-α mRNA tend to be higher in brains of patients with severe dementia.

Differential diagnosis of AIDS dementia includes opportunistic infections of the central nervous system like toxoplasmosis, Cryptococcal meningitis, tubercular meningitis, Cytomegalovirus encephalitis, and neurosyphilis; Occasionally, progressive multifocal leukoencephalopathy and primary lymphoma of the central nervous system also enter into the differential diagnosis. The diagnostic work up includes neuroimaging to exclude brain abscess, progressive multifocal lenkoencephalopathy and lymphomas, as well as analysis of cerebrospinal fluid to exclude opportunistic infections. CSF pleocytosis is seen in less than 10% cases of HIV dementia. Magnetic Resonance imaging of brain reveals cortical atrophy and hyperintense signals on T2-weighted images from subcortical white matter. These hyperintense signals from white matter regions on T2WI are believed to represent dysfunction of blood-brain barrier. Quantitative MRI reveals loss of Volume in the region of the caudate nucleus. Positron emission tomography (PET) reveals subcortical hypermetabolism in the early stages of HIV dementia which later progresses to cortical and subcortical hypometabolism. Normalization of PET abnormalities has been documented after administration of antiretrovirals. Magnetic resonance spectroscopy (MRS) is most sensitive as compared to other neuroimaging techniques. MRS shows a reduction in N-acetyl aspartate (NAA) and elevation in choline-containing compounds relative to creatinine. The technique may also help in predicting neurological progression.

Clinical features of HIV dementia may be grouped under two headings :- a) Early HIV dementia characterized by forgetfullness, lack of concentration, loss of libido, apathy and social withdrawal. It may be accompanied by errors made in a task involving antisaccadic eye movements. The neuropsychological features of early HIV dementia reflect predominant subcortical involvement characterized by i) memory loss selective for impaired retrieval ii) psychomotor retardation iii) impaired manipulation of acquired knowledge. b) Late HIV dementia presents with progressive cognitive decline, mutism, impaired insight, hallucinations, decorticate posturing, myoclonus, seizures and spastic weakness. Price and Brew devised a staging system for HIV dementia. Thus a patient with subtle neurocognitive impairment without frank dementia will be categorized as Memorial Sloan Kettering 0.5 [MSK 0.5] and a patient with severe dementia as MSK3. On the other hand, a patient with subtle neurocognitive impairment with myelopathy shall be categorized as MSKI.

The course of HIV dementia is invariably poor, with an average life span of three to six months from the onsetof symptoms.

Main stay of HIV dementia is Zidovudine which is administered in an initial high dose of 1 gram daily. Response is better in Zidovudine naive patients. Erythopoietin (50-100 units per Kg of body weight twice weekly) should be given if anaemia develops. Neutropenia can be taken care of by G-colony stimulating factor [G-CSF].

Cognitive improvement may be seen within four weeks after initiation of therapy. For Zidovudine resistant cases, didanosine (ddl), Zalcitabine (ddc) or stavudine (d4T) may be substituted. In recalcitrant cases, experimental drugs like nimodipine, pentoxifylline and memantine may be tried. Pentoxifylline reduces TNF-α activity, nimodipine decreases gp120 mediated neurotoxicity, and memantine prevents NMDA receptor mediated neuronal damage. Peptide T, a synthesised octapeptide is believed to block gp120 and TNF-α induced neurotoxicity.

Dr. Parvaiz Ahmed Shah M.D; D.M
Consultant Neurologist,
Prof. Abdul Nafee M.D.
Department of Medicine,
Govt. Medical College, Srinagar.
Kashmir. India 190 009

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The Current Postgraduate Curriculum - "Need For Improvement"

JK Practitioner 1996; 3:2 75-76

With the globally adopted social goal of "Health For All By 2000 AD" gaining impetus, time is ripe for the medical schools to review and re-evaluate their existing systems of postgraduate training and education. HEA is a blatant challenge which calls for the most proficient managerial expertise for application of available medical knowledge in the community. Planning of educational objectives for postgraduate training will necessarily be based on the local health needs and national resources, and the ultimate acid test is their appropriateness in terms of financial and human resources on the one hand and the community's felt and unfelt needs on the other.

With a polotical commitment of all governments to provide the maximum possible health care for the entire poulation, the responsibility of community health providers is bound to increase, with an inevitable need for streamlining the education and training of postgraduates. The medical schools of developed world have been quick enough to heed the call of the new social compulsions. Most developed nations have adopted the model of increasing emphasis on the preventive and promotive aspects of health care delivery systems. In fact, in the Scandinavias the public health aspect is the pivot of medical teaching and health provision. The developing countries, that should have been the torch-bearers of the welcome change, unfortunately lag far behind. Very little changes, if any, have been done in the clinical and paraclinical branches to make the postgraduate training more peripheral, and consequently the 'ivory towers of diseases' remain as isolated as a French Castle.

Postgraduate training in public health (preventive medicine; community medicine) has been envisaged for training postgraduates for the establishment and management of modern health care systems besides their requirement as teachers. research workers and health educators. Though the courses are kept to vary from country to country and even within a country (depending on socioeconomic, demographic, religious and political situation as well as the customs, beliefs and attitudes of the community), essential components of a postgraduate degree in public health in any developing country will be similar. These must include certain basic sciences, social sciences, bioststatistics, deymography, epidemiology, family medicine and environmental sciences. Health administration and health education must form an integral part. Evidently a standard curriculum cannot be prescribed for all medical schools; the best approach being to establish a set of objectives based on local health needs and demands that the selected course must help the students to achieve.

The current curriculum of M.D. in SPM in our medical college is very close to an appropriate course. Most of our postgraduates come from the cadres that have already worked in rural areas for 2 years or more. Many of them pass their first year or so in the periphery where they are exposed to our rural field practice areas. The thesis is generally field-based and demands active research work in the periphery. After submission of the thesis, the student is required to work by rotation in microbiology, pathology, gynaecology and Obstetrics, and psychatry, and attend to the District Tuberculosis Centre (DTC), Leprosy Hospital, Food Laboratory, Blind Home and Beggar Home. This gives ample exposure to the postgraduate student.

Given the health situation in the country, the thesis topics allotted to the students should be need based. For this a syllabus should be devised at the national level pertaining to the topic of current intrest. There should be a free flow of researsh information amongst the sister institutions. Topics to postgraduate students should be allotted keeping in mind the disease pattern existing in the peripheries, and the result must be published in the reputed journals so as to ensure interaction through literature.

Keeping in view the vast area of the country and the geographical variations of disease and health-related events, the postgraduates should be given orientation and field exercises in different areas to make them accustomed with the epidemiological pattern of various endemic diseases.

To improve the quality of teaching it becomes imperative that the faculty members in the department should be reoriented from time to time so that they can update their knowledge and skills for the betterment of postgraduate training. To strengthen the teaching at undergraduate as well as postgraguate level newer techniques need to be introduced leaving the era of chalk and black-board behind.

Collaborations and active participation of other departments of medicine is mandatory to effective health provision at the peripheral level. ROME scheme (Reorientation of Medical Education) was started in 1977 consequent on the recommendations of the Medical Education and Support Manpower (popularly known as the Srivastava Committee), with the objective of involvement of medical colleges in community health programmes so as to develop a comprehensive health referral complex. Some medical Colleges have shown reservation about its implementation. It is an ideal scheme, as it gives an insight into the rural settings to both the undergraduate and the postgraduate students as well as their teachers. Interaction is most benificial for postgraduates on the clinical side who otherwise would never have an opportunity to work in distant rural areas. Serious attempts should be made to revive it and its implementation made mandatory.

The department of public health provides a unique opportunity of ensuring intersectoral coordination. Specialists in different associated departments should also be involved in the teaching of postgraduates in community medicine.

Dr. Abdul Rouf,
Prof. & Head
Deptt. of S&P Medicine,
Medical College Srinagar
Kashmir 190 010.

Guest Article:

"Percutaneous Cardiopulmonary Bypass Support in High Risk Intervention

Fayaz A. Shawl *

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Since Dr. Andreas Gruentzig's initial description of this innovative thearapeuth technique, percutaneous transluminal coronary angioplasty (PTCA) has revolutionized the practice of invasive cardiology. Initially envisioned by its creator as an alternative to coronary bypass surgery for a selected subgroup of patients with single vessel coronary artery disease, PTCA is now considered a legitimate approach for multi-vessel disease, unstable angina, acute myocardial infarction, and in symptomatic patients who have previously undergone coronary bypass surgery.[25]

Despite this dramatic increase in the use of PTCA doubtlessly due to technological advances as well as increased opertation expence, there remain certain limitations, eg. restenosis, old total occlusions, diffuse disease, and an abrupt vessel closure. Abrupt closure occurs in approximately 5% of patients undergoing elective PTCA[67] and can resultin myocardial infarction, hemodynamic collapse, necessaty for emergency bypass surgery, and death.[8] Thus patients at high risk for the development of hemodynamic collapse in the event of abrupt closure are usually not considered for PTCA because they might not survive sufficiently long enough to undergo emergency bypass surgery. Subsets of patients considered at "high risk" generally have poor left ventricular function and/or a target vessel(s) supplying greater than one half of the viable myocardium. Such patients may not even tolerate transient myocardial ischemia during balloon inflation. PTCA in this high risk subgroup has been accomplished using a prophylactic infromotic baloon pump[9]; however hemodynamic collapse occurs, particularly when the patient has no intrinsic rhythm, the intra-aortic balloon pump is not effective. On the other hand, cardiopulmonary bypass support even in the absence of cardiac function.

Kantor et al[10] revived 6 patients in cardiac arrest or cardiopulmonary bypass support system. These patients were unresponsive to conventional resuscitation methods including intra-aortic balloon pumping. Subsequently, Vogel et al[11] reported 3 patients in whome PTCA was performed using a prophylactic cardiopulmonary bypass support system. In both studies[10,11], the bypass cannulae used were placed by surgical cutdown, which is impractical in the catheterization loboratory, particularly in a hemodynamic crisis. Subsequently, the author[12] developed and introduced a percutaneous insertion technique for initiation of cardiopulmonary bypass support using initially 20Fr and now 18Fr cannulae. When percutaneous cardiopulmonary by pass support is used on an elective basis in high risk PTCA, the term "supported angioplasty"has been adopted[11,13,14]. Percutaneous cardiopulmonary by pass support (PCPS) has also been used to support patients in cardiogenic shock and cardiac arrest.[15-17]

Technique of Percutaneous Cardio-pulmonary Bypass Support (PCPS)

The procedure is performed in the cardiac catherization laboratory with the patient awake. When PCPS is instituted electively in high risk patients, iliofemoral angiography is performed first in order to assess the suitability of the iliofemoral system for insertion .od bypass cannulae. This is accomplished by using 7Fr pigtail catheter introduced through the right femoral artery. The tip is positioned just above the bifurication of the descending aorta and 25cc of contrast is power injected at 10cc/sec. If the left iliofemoral system reveals acceptable anatomy, a awan ganz thermodilution eatheter is introduced through the right femoral vein for hemodynamic measurements. The 7Fr pigtail catheter is removed, and a standard 8Fr angioplasty sheath is introduced. The cardiopulmonary bypass support annulae are inserted through the left iliofemoral system reveals unacceptable anatomy, in which case the contralateral femoral vessels are used. Venous and arterial access to the left femoral system is obtained and standard 8Fr sheaths are left in place. Heparinization is accomplished using 250-300 units/kg of body weight to obtainan activated clotting time >400 seconds. Then, a flexible 0/038

Director Interventional Cardiology ,
Washington Adventist Hospital
Clinical Associate Prof. of Medicine (Cardiology)
George Washington University Hospital. (USA).

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inch J tip guidewire is introduced through the 8Fr arterial sheath. Keeping the guidewire in place, the standard 8F sheath is removed and replaced by an 8Fr long diator (C.R. Bard, Inc., Billeria, MA). Leaving the *Fr long diator in place, the flexible guidewire is replaced by a stiff 0.038 inch flexible tip guidewire (C.R.Bard, Inc.), making sure ti leave the flexible tip of the guidewire above the level of the disphragm. Holding the stiff guidewire in place, the long 8Fr dilator is removed and the vessel is dilated with a 12Fr and then a 14Fr dilator by passing each over the stiff guidewire with a rotary motion. The skin at the puncture site is incised for 2 to 3 mm to facilitate insertion of 18Fr cannule (in our initial series, 20F cannula were used). After placement of the cannulae in the femoral artery, the guidewire and the introducer are removed and the cannula tubing is closed using Robert's clamp. Using a similar approach, an 18F venous cannula is inserted in the left femoral vein. The tip of the venous cannula is positioned under fluoroscopy just above the junction of the interior vena cava and the right atrium.

When PCPS is instituted in the setting of cardiac arrest, iliofemoral angiography is not performed. In patients who sustain a cardiac arrest in the catheterization laboratory, cathetere are exchanged (following further dilation) for bypass cannulae.

The cardiopulmonary bypass support system's perfusion circuit is primed with 1,400 ml of Normosol by the perfusionist while the percutaneous cannulae are being inserted. The bypass cannulae are then connected to the portable cardiopulmonary support system (CTS C.R. Bard, Inc.).

When PCPS is used to support high risk angioplasty, cardiopulmonary support is initiated at a flow rate of 2 L/min and, if necessary, increased by increments of 0.5 L/min if pulmonary wedge pressure is > 5mm Hg, if there are ischemic electrocardiographic changes, or if the patient has chest pain during balloon inflation. Most patients require between 2.5 to 3.5 L/min of flow. PTCA is then performed through the contralateral femoral artery in the standard manner. Once succesful angioplasty has been performed cardiopulmonary support is gradually weaned in a manner of 2 to 3 minutes and then the cannulae are clamped. While reducing the flow rate during termination od cardiopulmonary bypass, volume infusion is usuallt required in order to raise the pulmonary capillary wedgepressure (or pulmonary artery diastolic pressure) to 8 to 10 mm Hg, or at least to the pre-bypass level. In most patients, volume infusion of 200 to 700 cc are required to obtain adequate blood pressure prior to termination of cardiopulmonary support. In out initial series, the bypass cannulae were removed in the catheterization laboratory when the patient appeared to be clinically stable. An external clamp was used for hemostasis after the patient was placed on a special stretcher. This method required prolonged clamping (8 to 16 hours). The method has been modified so that now cannulae are removed 6 hours after the last dose of heparin. The patient is taken to the coronary care unit for observation after the procedure, and the cannulae are removed there. Following the removal, manual compression is applied just above the insertion site for about 15 to 20 minutes and then a modified compression clamp (Compressar, Instromedix, Hillsboro, OR) is applied. With the current approach of cannulae removal and clamping, the total clamp time for complete hemostasis has been reduced to less than 4 hours

When PCPS is instituted in an emergency situation, such as cardiogenic shock or cardiac arrest, an estimate of the blood flow requirement can be made based on the patient's body surface area or the body weight (2.2 to 2.4 L/mm per square meter or 50 to 60 ml/kg of body weight). In order to assess the adequacy of tissue perfusion, frequent measurements of mixed venous oxygen saturation are made and it should be kept above 65%. In emergency cases, the patient is left on bypass support until the underlying problem is corrected. If emergency coronary bypass surgery is indicated, the patient is transported to the operating room while on bypass.

Hemodynamics:

The currently available portable CPS system (C.R. Bard, Inc.) provides non-pulsatile flow. This system differs significantly from the traditional heart-lung machine in that it allows a flow rate up to 6 liters per minute using 18F-20F cannulae. This compact system consists of a centrifugal, non-occlusive pump, polypropylene hollow fibre membrane oxygenator and heat exchanger. Using a Vortex pump, blood is actively aspirated from the right atrium, which after passing through the heat exchanger and membrane oxygenator, is finally pumped back into the distal aorta. Using 20Fr cannulae, a flow of 5 to 6 L/min can be achieved, while with 18Fr cannulae a flow up to 5 L/min can be achieved, provided the patient is well hydrated prior to the institution of cardiopulmonary bypass. After the initiation of bypass support, there is a rapid fall in the filling pressures manifested by a reduction in pulmonary capillary wedge (or pulmonary artery diastolic) pressure to approximately 0 to 5 mm Hg. There is also a reduction in the systolic arterial pressure with slight increase in the diastolic pressure, the later feature known to be present with a non-pulsatile flow. Most patients experience a warm sensation, most likely due to vasodilation. The urinary output is increased suggesting increased rental perfusion. Echocardiography performed on patients while on PCPS has demonstrated reduction in the left atrial and left ventricular dimentions. These obsesvations, together with the reduction in the preload and afterload, suggest that PCPS reduces myocardial oxygen demand which may, in part, be responsible for the absence of angina or electrocardiographic charges duting balloon inflation in most patients undergoing supported angioplasty. However, segmental wall motion clearly decreases during balloon inflation indicating ischemia distal to the occlusion.

PCPS can be used for up to 6 hours without any evidence of hemolysis or thrombocytopenia, but cannot be used for prolonged periods, especially when used in the setting of cardiac arrest or cardiogenic shock.

Clinical Experience:

We have used PCPS both emergently, and electively to support patients undergoing high risk PTCA and valvuloplasty, as well as patients in cardiogenic shock, or cardiac arrest[13-17,19]. All procedures were performed in the catheterization laboratory. The capability of PCPS to provide hemodynamic support in patients with cardiac arrest has been impressive. Prior to cardiac arrest, patients were either in cardiogenic shock or had experience abrupt closure during PTCA[17]. PCPS permitted unhurried performance of complex angloplasty or transfer of the patient, in stable condition for coronarybypass surgery. Seven patients who had failed advanced cardiac life support have been supported with PCPS, some of them remained awake and responsive to verbal commands while their rhythm was ventricular fibrillation. It was of intrest to note that 4 of the 5 patients who underwent revascularization (PTCA or bypass surgery) after hemodynamic stability was achieved, survived. Survival appeared to be dependent upon whether the underlying lesions were amenable to definitive therapy. Two of our patients, whose vessels were unsuitable for revascularization, expired. The only patient who died after revascularization had a 45 minute delay prior to the institution of PCPS, which suggests that the early use of PCPS may be necessary for a successful outcome. The same conclusion were drawn in a recent study by Overlie et al.[18]

PCPS was also instituted on an emergency basis in 8 patients in cardiogenic shock due to acute myocardial infarction.[16] PCPS was instituted at a mean of 106 minutes after the onset of cardiac shock. Four patients had a history of previous myocardial infarction and 2 were in cardiac arrest. Hemodynamic stability was achieved in all 8 patients at a flow rate of 3.2 to 5.2 L/min and, 2 patients in cardiac arrest regained consciousness while still asystolic or in ventricular fibrillation. One patient had anatomy unsuitable for either PTCA or bypass surgery, could not be weaned from PCPS, and expired. The remaining 7 patients underwent successful PTCA in 15 of the 16 lesions attempted and were weaned off PCPS. The total bypass time in this age group was 44 to 120 minutes (mean 66). At a mean follow up of 6 months, all patients are alive, 6 are asymptomatic and 2 have Class II angina.

Most patients underwent PCPS prophylactically during high risk PTCA. Patients were considered for supported angioplasty if they had severe (Canadian Cardiovascular Society Class III or IV) or unstable angina with poor left ventricular function (ejection fravtion of (25%) and/or vessel(s) to be dialated supplying greater than one half of the remaining viable myocardium. Some of the patients who underwent PCPS were inoperable because of severely derpessed left ventricular function, multiple prior bypass surgeries or severe systemic illness. Of the first 75 consecutive elective patients[19], 30 had Class III, 26 had Class IV and 19 had unstable angina. There was a history of myocardial infarction in 67 (89%), prior bypass surgery in 21 (28%) and congestive heart failure in 27 (36%). Forty-six (90%) had impaired left ventricular function (mean ejection fraction 31%), and 30 (40%) had ejection fraction of 25% or less. Twentyfour were considered inoperable by the local or referring institutions. PTCA was successful in 154 of the 158 lesions attempted with the culprit vesel successfully dilated in all. Dilatation of the patient's only remaining vessel was performed successfully in 23. Inflation times up to 10 minutes were well tolerated. Chest pain and ischemic electrocardiographic changes were noted in less than 20% during balloon inflation. Average bypass time was 37 minutes. No deaths occured during the procedure and no patient required emergency bypass surgery. Hemostasis was achieved by external clamp compression in 73. There were 3 in-hospital deaths unrelated to bypass. In this initial group of 75 patients, complication due to PCPS occured at the cannulae entry site in 13%, which included pseudoaneurysm of femoral artery, necessary for repair of femoral artery, embolus and femoral neuropathy. However, with experience and refinement of technique, local complications have been virtually eliminated (none in the last 32 cases). Also, necessary for blood transfusion is currently less than 3%.

Conclusion.

The development of percutaneous cardiopulmonary bypass support using the femoral vessels provides an additional and simple technique to support patients undergoing high risk interventional procedures in the cardiac catheterization loboratory. Our initial experience suggests that supported angioplasty can be sucsessfully performed in "high risk" patients with acceptable morbidity and mortality. PCPS can facilitate emergency bypass) in the setting of cardiac arrest or cardiogenic shock. The hemodynamic stability achieved during supported angioplasty not only permits PTCA to be performed in such high risk patients, but also reduces operator anxiety about arrhythmias and hemodynamic instability. Also, in the event of abrupt closure during supported angioplasty, emergency surgical revascularization, repeat dilation or event stent placement can be performed safely. Since PCPS produces substantial unloading of the left ventricle, the risk of myocardial infraction may be minimized. However, echocardiographic data demonstrate that ischemia does occur during balloon inflation, inspite of the unloading that occurs. This suggests that PCPS may not prevent a myocardium distal to the occlusion using a separate perfusion system is under investigation and may be beneficial under these circumstances.

Further studies will be necessary to define the exact role of PCPS, as well as the role of adjunstive therapy with techniques such as antegrade myocardial perfusion catheters[20,21], coronary sinus retroperfusion[22,23] and relative hypothermia for limiting myocardial injury. The role of PCPS as a bridge to other left ventricular assist devices should be explored.

Although the ultimate role of PCPS remains to be defined, I believe this technique will make a significant contribution to the interventionalist's armamentarium and extend the availability of PTCA to patients that were hitherto inaccessible.

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Clinical Review:

"Diet and Colorectal Cancer"

*Nazir Ahmed Wani, **Omar Javed Shah, **Tajamul Fazili

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The problem of colorectal cancer in developing countries is a significant one. By the age of 75 years, about 5% of the population in the united states will develop colon cancer and another 1% to 2% will develop rectal cancer (1) In the united states, Canada, U.K and most other Western Countries colorectal cancer is the second major cause of death from cancer. For several decades a cure rate for this cancer has remained static at 50% (2). The tremendous interest in investigating the causative factors for colorectal cancer in the hope that this knowledge would permit cancer prevention should not come as a surprise. What remains disappointing is the accumulation of a massive body of information has not yielded definite results. Even today the cause are not known with certainity.

It seems that environmental factors have definite role in the etiology of the diasease, for there is a difference in the incidence of colo rectal cancer between countries and a change in prevelance rates among migrants. What has emerged as one of the most plausible etiological factors is diet. An intuitive appeal to the dietary hypothesis is rendered by the direct contact that takes place between dietary constituents and the organ at risk. Epidemiologic studies and animal experiments are the primary source from which the data that include dietary factors in the etiology of colo rectal cancer come from.

Only the dietary contituents of the many factors studied, particularly the proportions of animal protein and fat, have been shownr to correlate well with the known geographical variations of the colonic cancer. Thus, diet has come to occupy such an important place in the development of this disease and has given us hope to believe that it can be prevented. Over the past twenty years three principle theories have been advanced to explain the etiology of this tumor. George Oettle, a South African, in 1960 developed the first of them which was popularised later by Denis Burkitt. This may called fibre hypothesis. Wynder in 1965 outlined the second one at a symposium in New York which has been termed the animal fats hypothesis. Bjelke at the university of Minnesota in1973 developed the third theory. Although more recent it is less know then the other two and is called the vegetabler hypothesis. Experimental and Epodemiological work support each of these these, and though seem to be mutually exclesive at first sight,the ultimate truth may well be shown to encompass all three.

Fibre Hypothesis:

This hypothesis takes roots from the dual observation that in South Africa Bantus the incidence of large bowel cancer is low and the stool they produce is bulkier than their white counterparts. High intake of dietary fibre was thought to be responsible for the bulkier stool. A simple and logical theory relating low dietary fibre to the development of large bowel cancer was formulated by Burkitt in 1977 (3). He prepounded that a reduction in the intestinal transit time which reduces the exposure of the gut wall to potential carcinogen is due to the high fibre intake. In addition, the probability for bowel bacteria to produce carcinogens is greatly reduced because a dilution of these occurs (4), when the bulk of the stool increases, while condiderable popularity has been enjoyed by this hypothesis, it contains several contridictions. That high fibre intake consistantly shortens transit time cannot be proved conclusively. Some studies have registered a decreased transit time (5) while others have shown no effect at all (6). The most potent evidence illustrates that natural fibrem, particularly wheat bran, normalizes transit time. Thus it is accelerated in those with slowed transit times, slowed in those with rapid transit times and left as it is in those with moderate transit time (7,8). That a simple inverse corrolation exists between transit time and bowel cancer is suggested by the evidence that the Japanese in Hawai havea lower colon cancer incidence than the Americans living there. Yet they have, if anything, a slower transit time (9). Burkitt's epidemilogic studies attempted to look at what he believed to be comparable groups in Africa and England. Although the Africans exhibited a shorter transit time than the English, yet he did not question the validity of

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* Prof & Head Deptt. of Surgery
** Lect. Deptt. of General Surgery
Sher-i-Kashmir Institute of Medical Sciences,
Soura, Srinagar Kashmir.

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extrapolating these findings, which were in healthy people of different culture and race, to people with colon cancer. At no time did he compare bowel cancer patients with controls. No correlation between dietary fibre and the geographical incidence of large bowel cancer was found in two major international surveys conducted (10,11). The final conclusion that comes through is that Burkitts fibre hypothesis has gained considerable vogue not because of any intrinisic scientific merit but because of undue publicity accorded to it.

Animal Fats Hypothesis:

This hypothesis links the development of bowel cancer to high animal fat intake. The principle of this theory have been advocated by Wynder and Reddy. Wynder puts forth that increases in dietary fats are accompanied by increases in bile acids, neutral steriods and fecal bacteria which metabolize the fact substances into carcinogen products, possibly related to methyl cholanthren (12). This initial evidence to support his theory come from a study of twenty one countries wherein he demonstrated that an increase in per capita intake of animal fats increased the morality from colon cancer. Other workers (10,11) have substantiated this finding. Empliying similar methodology Enstrom produced results which were contrary to the earlier findings. He showed that the upper socio economic groups in USA were characterised by a higher ingestion of animal fats and proteins yet failed to demonstrate any increased propensity towards the development of colon cancer (13) . He demonstrated that a higher intake of beef over the past 40 years contributed only to a marginal increase in the incidence of bowel cancer over the same period . One of the most important factors, contributing to the high incidence of colon cancer in countries like Argentina and Uruguay may be the high consumption of beef in these countries. Not surprisingly enough with the decrease in the intake of beef in Australia between 1955 to 1965, the morality from colon cancer dropped by 19% in the same period (14). On the contrary the Mormon population in Utah that has registered a per capita increase in beef consumption over the past twenty five years does not show any increase in their relatively low incidence of colonic cancer (15). The animal fat hypothesis is supported by two studies whereas other studies have founf no difference in the ingestion of meats by cases and controls. Wynder and Reddy have substantiated their hypothesis by demonstrating great fecal excretion of bile acid and neutral steroids in Omnivorous people compared to those who eat little or no meat (16,17). Additionally they discovered a greater concentration of these substances in the faces of cancer patients than controls (18), plus a greater enzymatic activity of the fecal flora in cancer patents. Furthermore they have shown that rats fed on either vegetable oil or beef fat and trreated with Dimethyl hydrazine were more likely to develop colonic tumours than animals on a controlled diet (19) .

The Carcinogenic activity of Bile acids theoretically relates to structural simlarity to carcinogens like methyl cholanthrene and possibility of bacterial conversion of dexycholic acid to this or related compounds. Drasar et al and Hill (20,21) reported clostridium paraputrrificum numerously present in faces of colon cancer patients than as compared to controls. This organism in known to dehydrogenate the steroid nucleus - an effect that would be important in development of compounds with structures similar to those of known carcinogens. Although this theory has a firmer scientific foundation than Burkitt's fibre hypothesis but there remain may hurdles before this theory is uniformaly accepted.

Vegetable Hypothesis:

The hypothesis which has come to forefront recently has shown considerable promise. It was Eric Bjelke who compared patients of corectal cancer with non - malignant diseased patients and found less frequent ingestion of vegetable and cabbage in particular. A study conducted by Modan et al (22) showed that higher ingestion of fibre lowered the risk of colonic cancer. This study supports the vegetable hypothesis for the group of fibre containing foods which they studied included many vegetables. The highlight of this study was that patients with colonic cancer eat less cabbage than controls. Similar differences were observed for other cruciferous vegetables like Kohlrabi, Sauerkraut,Turnips and cauliflower.

The fact that there is enough evidence to suggest that animal fact and meat promote a high risk of colonic cancer may be an indicator that vegetables will lower the risk, for a reciprocal relationship exists between these constituents of diet.

Before the vegetable hypothesis may be validated, much work needs to be done both in animals as well in humans. However, we cannot overlook the fact that almost all the research in this direction has produced congruent results

Miscelleneous Factors:

Several other dietary components have been implicated in the etiology of colonic cancer from time to time. To illustrate we have an example of Yudkin (23) and Cleave (24) who attempted to relate the development of colonic cancer to the high sugar consumption of western people. However, they failed to support their findings with convincing evidence. Experimentally, naturally occuring substances as vitamin C, vitamin E, Selenium and Calcium have been found to protect animals against the induction of tumours. It cannot be considered to apply to haman population unless epidemiological evidence accumulates in favour of this theory in relation to the human population. Colorectal cancer has a multi -factorial etiology though there is a strong evidence suggesting a role for dietary factors in the cauisality of this disease. The evidence points overwhelmingly to the key role of environmental factors and diet in particular. Ironically, though Burkitt's fibre hypothesis is perhaps the most well know it is the least established scientifically, while none of the theories provide all the answers, there is a strong evidence suggesting that a high animal fat/protein intake inhance the formation of colonic cancer, whereas certain cruciferous vegetables lower the incidence.

Correspondance to:-
Prof. Nazir Ahmed Wani,
Bypass Crossing Hyderpora,
Srinagar-Kashmir (India) 190 014

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Clinical Review:

"Approaches to Child Health"

*** G. M. Dar**

JK Practitioner 1996; 3:2 85-88

Hospital Approach:

Traditionally hospitals enjoy a prestigious place in the health system of any country and are regarded as the 'be all' and the 'end all' of all that is connected with health and well-being.

In industrialized countries, where social problems like poverty, ignorance, illiteracy, unemployment and under employment, and the environmental issues like housing, water supply and sanitation etc. are being addressed routinely and have, by and large, been taken care of, the major child killers of the developing world like malnutrition, acute respiratory infection and diarrhoeal disease, have lost their significance. The hospitals, in the developed world, are essentially reserved for referral cases in need of specialized care and advanced levels of management.

On the contrary, in the developing countries, where child population is threatened by the mutually reinforcing problems of infection, malnutrition and uncontrolled reproduction, a primary health care approach is the only strategy that can attack these problems at the grass-roots level. An imported hospital model, based on 'adoption' rather than 'adaptation' is irrelevant and out of place. What is really needed, is that hospitals should be sensitive to the existing health care services of the locality. Hospitals should organize outreach activities and assume the primary health care responsibilities of the neighbourhood population besides offering specialized care to cases referred by the primary health organisation.

Nothing could be more damaging to child health than the establishment of a western-modeled hospital system, out of tune with the health care needs of the child population and in perfect isolation from the primary health care effort, though consuming the lion's share of the health budget. In the developing countries, in particular, the 'ivory tower' medicine should yield place to an open hospital system with multiple functional linkages with primary health programme activities especially in the area of maternal and child health.

Primary Care Approach:

'Primary health care is a practical approach to making essential health care accessible to individuals and families in the community in an acceptable and affordable way and with their full participation. This approach has evolved over the years, partly in the light of experience, positive and negative, gained in basic health services in a number of countries. But it means much more than the mere extension of basic health services. Its shape is determined by social goals, such as the improvement of the quality of life and maximum health benefits to the greatest number ; and these goals are attained by social means, such as the acceptance of greater responsibility for health by communities and individuals and their active participation in attaining it.' (Alma Ata, 1978).

Primary health care approach has revolutionalized the health care delivery system of the countries, all over the world. It has not replaced the basic health approach, but has added to its dimensions vertically as well as horizontally. In other words, primary health care is an extension, as well as, an expansion of basic health services. By extension it makes essential health care accessible to families and individuals within those families, and by expansion, it involves all the health and health-related sectors in the process of primary health care. In practical terms, extension has been affected by involving community level workers and the expansion has been achieved by promoting intersectoral co-ordination. The absence of intersectoral approach can have detrimental effect on community health. A rational multisectoral approach can help eliminate undesirable counter-productive processes and reinforce mutually supportive activities contributing to general health and well-being of children along with others.

Primary health care has been accepted as the means for attainment of 'Health of All'. MCH care, which constitutes the core element of primary health care package, is provided by front-line health workers. In the system obtained in this country, child health care is delivered by female health workers and traditional birth attendants (during perinatal period) under the supervision of female

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Professor & Head Social & Preventive Medicine,
Jehlum Valley College Srinagar Kashmir
(Ex. Professor SPM, SKIMS Srinagar, Kashmir)

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health assistants. The involvement of female workers, in India, has immense sociocultural relevance and has improved the accessibility and acceptability of MCH services in rural communities. Primary health care approach is the mainstay for child health and the most effective means of managing major child health problems at the most appropriate level.

Risk Approach:

Risk approach is a cost-effective and result-oriented management device in health care. The approach is built on the identification of risk factors having a direct or indirect association with a disease process or a health problem; identification of the population under influence of these factors; and the institution of measures of risk intervention. While risk approach provides for basic care for all; It ensures greater care for those at greater risk, simultaneously facilitating rational distribution of resources and more complete health care coverage of population, at no extra cost.

Risk approach has a wide scope in primary health care, especially for identifying susceptible population groups, for planning health programmes, allocating resources and estimating manpower needs and their training requirements.

Risk approach is ingrained in primary child care. Gradation of children according to the levels of risk exposure provides the rationale for management of cases at primary level as well as for their referrals to the higher levels. The risk-factors that have been identified in infants and children are: 1) twins or low birth-weight; 2) breast feeding not established or insufficient; 3) single parent; 4) working mother; 5) failure to gain weight during three successive months; 6) loss of weight during two successive months; 7) history of death of more than two siblings during the two years of life; 8) history of acute episodes of illness such as gastroenteritis, measles, whooping cough; 9) birth weight below 70% of the reference value; 10) birth order 5 or more; 11) spacing less than 2 years; and 12) illness of parents/family members.

Identification of 'at risk' children in ICDS projects is based on weight-for-age cards having four reference lines. Children who lose weight or whose weight remains stationary or whose weight falls below the lines III and IV are at risk and need special care which may involve hospitalization. In the management of diarrhoeal diseases, children are graded into mild, moderate and severe degrees of dehydration. Children showing the evidence of severe degree dehydration have suffered 10% or more fluid loss and are at risk. They need immediate referral for intravenous rehydration.

Risk approach fits admirably into the primary health care system and facilitates a rational utilization of material resource and health manpower in a manner that does not lower the quality of health care.

Educational Approach:

For all practical purposes, the recipient of education on child health is the mother, the connecting link between the consumer and the provider. Educational investment in the mother is the best investment for the child, provided that it expresses itself in healthy child care practices. Health education must strive to motivate the mothers to fulfil their responsibilities towards breast-feeding, appropriate weaning, immunization, oral rehydration, personal hygiene, environmental sanitation, family planning and responsible parenting. It must dispel doubts and remove misconceptions, especially those lurking in the minds of ignorant women; it must counter the propaganda generated by unscrupulous elements and must warn the mothers against exploitation through malpractice.

Health education is not a passive process of informing a gathering of 'spectators' on various aspects of child health. It is a process of active community involvement. It should not be used as a last resort, nor should it be carried out in a vacuum. It should remain as an integral component of all the child health programmes and should be supported by adequate service complement to meet the demands that it generates.

'Health education must lead to a situation where people want to be healthy ; know how to attain health ; do what they can individually and collectively in maintaining health ; and seek help when needed' (WHO).

Public Health Approach:

Conventional public health measures like provision of potable water supply ; sanitary disposal of community and industrial wastes ; drainage and sewerage systems ; sanitation of food processing establishments ; and abatement of atmospheric pollution etc. add to the safety of the environment and protect the health of the population including infants and children. National disease intervention programmes directed at the control or eradication of major public health problems have a greater impact on population, especially on the valuable child segment. The public health programmes of relevance to child health in India, are those dealing with (a) infections, such as diarrhoeal diseases control programme, universal immunization programme, tuberculosis control programme etc.; (b) malnutrition, such as prevention of blindness programme, national goiter control programme, prevention of food adulteration programme etc. ; and (c) uncontrolled reproduction such as family welfare programme, post-partum programme etc. While the environmental measures are essentially indirect and impersonal and mainly addressed to prevention of diseases and promotion of health, the disease intervention programmes are direct and personal, dealing with prevention and/or treatment of specific health problems. When both kinds of programmes are executed simultaneously, a synergistic effect is generated, making further improvements to the health and well-being of population including children.

Regulatory Approach:

Regulatory approach to child health which usually takes the form of health legislations may deal with (a) public response to important medical events such as registration of births and deaths or notification of communicable diseases; (b) licensing of health personnel doctors, nurses, midwives etc. or institutions such as hospitals, nursing homes, day-care centers, creches etc.; (c) health care of children such as universal immunization, health screening on admission to schools etc.; (d) welfare of children such as abatement of child abuse, child neglect and various other forms of exploitation of children ; and (e) reproductive behaviour of parents like defining minimum age-limit for marriage, maximum family norm, legal requirements for termination of pregnancy etc.

Regulatory measures should be formulated after a broad survey of the problems and extensive consultation with the agencies, individuals and organisations directly or indirectly involved in the area to be brought under regulation. Conventionally legislations are brief and concise and can be applied only by following the rules and procedures usually framed along with the laws. Before introducing a legal measure, it is mandatory that all facilities necessary for conforming to the requirements of the legislation be available and accessible to the population and an effective administrative machinery be ready for vigilance and enforcement. A legislative instrument is only an adjunct to the basic health effort and cannot replace it in any manner.

Developmental Approach:

Development and health are interrelated ; while development promotes health, health contributes to further development. Development, to be meaningful, must permit people to lead economically productive and socially satisfying lives. A balanced socioeconomic development should be able to achieve social justice, poverty alleviation, higher standard of living and improved quality of life. Most importantly, it should be an instrument for bridging the gap between the 'haves' and the 'have nots'. This is possible only when economic development is linked to social goals and benefits of development are made accessible to the poorest of the poor. In the absence of social safeguards, development is destined to bypass the poverty segments and further increase the void between the rich and the poor.

A socioeconomic development based on social justice offers the most rational approach to child health. It removes the conditioning factors like poverty, illiteracy, unemployment, etc. associated with high levels of morbidity, malnutrition and mortality in children. Poor socioeconomic development arrests the individual development of young children; even a temporary deprivation during their growth period can result in permanent damage to their physical or mental make-up. This is the highest price that a nation can pay. The lapse of today can be a recurring loss for tomorrow, manifesting as, mounting debt repayments, falling export earnings, increasing food costs, falling family incomes, narrowing educational opportunities and shrinking health care facilities. Such a developmental reversal has taken place in some of the sub-Saharan African and Latin American countries, characterized by a progressive reduction in important economic indicators such as per capita GNP, debt repayment capacity and productivity of the labour. The industrialized world countries, dictating the global economic policies and strategies, are partly responsible for this crisis. Some of the Asian countries, specifically China, India, Sri Lanka etc., are progressing on the road to socioeconomic development, yet the majority of the world's poor are in Asia; the fruits of development do not reach those lying below the absolute poverty line who live in slums, shanties and neglected rural areas everywhere. It is very important that essential health and survival needs are reliably met, not just for the one billion or more who are the absolute poor of the world, but for the 2 billion people who will be constituting the poorest third of the world's population.

The 'seven sins' of development identified by UNICEF (The State of World's Children, 1989) are : (1) development without infrastructure; (2) development without participation; (3) development without women; (4) development without environment; (5) development without the poor; (6) development without the doable; and (7) development without mobilization. Elimination of these errors can go a long way in promoting a balanced socioeconomic development based on distributive justice and conducive to the health and survival of children.

Social and economic development should go hand in hand, for economic development is necessary for achieving social objectives and social development is essential for realizing economic goals. Child health is a social objective and development is the means to achieve that objective.

New Horizons in Non-Invasive Surgery:

"Extra Corporal Shock Wave Lithotripsy(ESWL)"

*** Muneer A. Khan,**

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Less than 5% of kidney stone are treated by open surgery in most Urological centers in the World. 96% of renal stone diseases is treated by a combination of E.S.W.L, (extra corporal shock wave lithotripst), PCNL (percutaneous nephrolithotripsy and URS (uretererenoscopy). About 60 to 70% of the renal and ureteric stones can be treated by ESWL alone.

The principal of ESWL centers on generation of hydraulic shock wave produced outside the body. The externally generated shock wave travels through the body without causing damage. because there is no difference in acoustic impedance between body tissues and water. However, provided the waves are correctly focussed at the tissue stone interface, partial reflection of waves occurs, ultimately causing short term mechanical stress and there fore a zone of destruction at the stone site. This focussing is accomplished by the specific design of the instrument.

An electric cell spark is generated between two electrodes placed under water. Thus creating a hydraulic shock wave. The wave is produced within a rotationally symmetric semi-ellipsoid at its F-I focal Point. The properties of Ellipse dictates that the waves are reflected off the elliptical wall and converge on a second focal point F-2. The patient is positioned on a bag of water, so that the kidney stone is located at the F-2 focal point. The stone is disintegrated by the shock wave traveling through the water bag and the patient's body on to the stone which is focussed at F-2. The positioning is accomplished by two plane X-ray system, whose axes intersect at the F-2 focus. Under Fluoroscopic guidance, the patient is moved into precisely correct position. A series of shock waves are then produced causing the stone to gradually disintegrate. The resultant small powder like fragments are subsequently passed with the patient's urine.

E.S.W.L. in Kashmir:

Off late we have acquired one of the World's most advanced Compact Lithotrypter (Tripter compact from Direx Medical System) in the Valley. This universal shock wave Lithotrypter meets a wide range of requirements for diagnostic evaluation and therapeutic treatment of kidney, ureteral and biliary stones.

The Digiscope-RX2 is a high quality imaging system which provides a mechanical combination of Fluoroscopic and Ultrasonic imaging, displayed on a high resolution monitor.

The incorporation of the Digiscope in the Tripter COMPACT architecture provides the possibility to combine fluoroscopic and ultrasonic imaging. Stone localization and patient positioning are performed with the aid of the computerized system. The ability to display side-by-side multiple images related to various phases of the ongoing treatment significantly improves the monitoring of the treatment.

From 6th December, 1995 sixteen patients with renal stones (volume 1-3 cm) have been treated on this system. The stone breakage has been achieved in fifteen patients (94%) in one or more sittings. The average shock waves per centimeter of stone has been 2000-2500 shock waves. The procedure is relatively painless, requiring sedation. There was no need of anesthesia in any of these patients.

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* Asst. Professor & Consultant Urologist,
Deptt. of Surgery, Govt. Medical College
Srinagar, Kashmir. India 190 009.

Photograph Showing EWSL Procedure.

The procedure was carried out as out-patient procedure. No patient needed overnight admission. Internal stenting was carried out in five of the sixteen patients (31%) with ureteric and large volume renal calculi.

Contraindications to ESWL:

There is no absolute contraindication to this procedure. Relative contraindications are:- bleeding disorders, pregnancy and obstructive uropathy. Obstructive uropathy and infection if present should be treated prior to ESWL. ESWL should be undertaken with extreme care in case of renal artery and other vascular calcification. In large volume stones ESWL should be combined with PCNL.

Sequelae of ESWL:

Skin bruising is frequently seen especially when the contact of the water containing bag with the body is not proper. Perinephric haematomas and hypertension has been reported in less than 5% of the patients.

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Original Articles:

Thyrotoxicosis: Management with Radio active Iodine (I-131) - (follow up study)

*Syed Mushtaq, *Shoukat H. Khan

Abstract:

Radioiodine treatment of thyrotoxicosis is an attractive modality in terms of cure rate, cost effectiveness and ease of administration. The only long term morbidity associated is hypothyroidism which is managed by exogenous thyroxin administration. In our study of 74 patients, we used a fixed dose of 10 mCi of I-131. A single dose was effective in controlling 87.5% of solitary toxic nodules, 74.2% of diffuse toxic goitres (Grave's disease) and 29.6% of toxic multinodular goitres. At the end of one year of following 10.9% of patients with Grave's disease developed hypothyroidism. Inspite of limited sample size and insufficient follow up, our results are on the expected lines and comparable to the results obtained in other Institutional studies: *JK Practitioner 1996; 3:2 90-93*

Introduction:

Thyrotoxicosis is one of the common endocrinopathies with profound metabolic derangements. It affects almost all vital systems of the body, and can lead to life threatening cardiac disease in the form of heart failure, arrythmias, ischaemic heart disease and arterial embolism [1,2]. Despite the rapid advances in diagnostic armamentarium, such as radio-immuno assay, ELISA, radioiodine uptake test and thyroid scan, a considerable difference of approach is seen among various physicians in the management of thyrotoxicosis. The classical approaches are antithyroid drugs, surgery and radioactive Iodine (I-131) ablation.

The antithyroid drugs do not produce a sustained long term remission. An early relapse or late recurrence of hyperthyroidism in at least 50% of patients when treatment is stopped even after prolonged course such as two years or more is reported [3,4,5]. Side effects of antithyroid drugs are uncommon. Allergic skin reactions are the commonest but the most serious is agranulocytosis and has been reported in 0.2-0.3% of patients [6]. The problem gets compounded by inadequate patient compliance and cost of treatment, especially in the third world countries.

Surgery (Subtotal thyroidectomy) offers higher chance of cure in a much shorter time but requires admission, an experienced thyroid surgeon and the patient's fitness and willingness to undergo surgery. Complications include haemorrhage, recurrent laryngeal nerve palsy and transient or permanent hypoparathyroidism. The morbidity ranges between 0.2 - 1%. Substantial number of elderly patients are poor risk due to cardiovascular complications of thyrotoxicosis and other associated medical problems. Cost and ease of administration is least favourable in this type of treatment. It is now used only for patients with very large goitres where there is suspicion of malignancy [6].

Since the introduction of radioactive iodine (I-131) in 1941 it has been widely accepted as a very useful therapy for thyrotoxicosis [7]. In the past, much caution was taken in choosing the patients for I-131 therapy, owing to fear of radiation induced cancer or genetic complications. There has been no evidence so far of an increased risk of thyroid or other malignancies or of genetic complications in patients who have been followed up for 20 years or more after I-131 therapy [6,8-10]. The therapeutic use of 1-131 in hyperthyroid children is claimed to be safe but has not yet received widespread acceptance in view of limited duration of follow up. Now a days the use of radioiodine is more liberal because of being very effective, safe, easy to administer and economically cheap. It is used both as a primary treatment and for relapse after medical therapy. Radioactive iodine provides the highest rate of cure, approaching almost 100%. The effective dose is variable and more than one treatment dose may be necessary [6,11].

In our study, 74 patients with thyrotoxicosis were administered I-131. These patietns were put on follow-up to evaluate their clinical response and study the overall behaviour of their disease process.

Material and Method:

Patients were referred to the department of Nuclear Medicine mostly by the Endocrinology

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*Department of Nuclear Medicine
Institute of Medical Sciences, Srinagar.

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department of SKIMS and some other hospitals of Kashmir. Age and sex distribution of these patients is shown (TableI). All the patients were asked detailed history and examined clinically. Lactation and pregnancy was ruled out in all female patients. A base line ECG was done in all the patients. Thyroid gland size along with the size of any nodules was estimated manually. T3, T4, TSH levels, Radioactive iodine uptake and thyroid scans were obtained in all the patients. The patients were categorized as diffuse toxic goitre(Grave's disease), solitary toxic nodule and toxic multinodular goitre (Table-I).

After confirming the diagnosis a fixed dose of 10 mCi (370 MBq) of I-131 was administered orally. Patients with severe clinical symptoms were prescribed beta blockers (Propranolol 40 mg BID). In addition some patients were prescribed anti-thyroid drugs (Neomercazole 20-40 mg in divided doses) for about one week after the first therapeutic dose of I-131, till it had its desired effect which was usually 4-6 weeks.

The patients were followed up at regular intervals of 6 weeks and 3 months. At 3 months post I-131 therapy patients were subjected to T3, T4, and TSH radio-immunoassays (RIA). Thyroid scan was also done in most of the patients. Patients rendered euthyroid were kept on follow up at 3 monthly intervals for first year and then 6 monthly for next year. Patients who persistently had thyrotoxicoxs after the first therapeutic dose of I-131 were administered a second dose of 10 mCi of I-131. They were followed up again at 6 weeks and three months.

Eight patients of Grave's disease were found to be hypothyroid by one year. They were put on substitution thyroxine (0.1-0.3 mg daily). They were asked to attend regular follow up at 3 month intervals. The T3, T4 and TSH estimations were carried out and dose of thyroxine was adjusted accordingly.

Results:

The majority 38/74 (51.4%) of thyrotoxic patients belonged to age group 40-55 years, followed by 24/74 (32.4%) and 12/74 (16.2%) in age groups of 25-40 years and 50-70 years respectively. Diffuse toxic goitre accounted for 31/74 (41.9%) of total cases with female to male ratio of 2:1. Toxic multinodular goitre was present in 24/74 (36.5%) of cases with female to male ratio of 4:1. Solitary toxic nodule accounted for 16/74 (21.6%) of cases, all of them females (Table I).

The mean values of T3 and T4 levels in our patients prior to I-131 therapy are depicted in Table II. The 2 hours and 24 hours uptakes with 25 microcuries of oral I-131 were uniformly raised irrespective of type of thyrotoxicosis (Table III).

In Grave's disease 74.2% of patients were clinically euthyroid with their T3 and T4 levels within normal limits 3 months after the Ist dose of 10 mCi of I-131. The remaining 25.8% required a second dose of I-131 and were euthyroid within 3 months of administration of second dose. 87.5% percent of patients with solitary toxic nodule became euthyroid within three months of single dose of I-131 and only 12.5% required a second dose. Out of toxic multinodular goitres only 29.6% responded to first dose of I-131, 22.2% responded to second dose and 25.9% had persistent toxicity after second dose. 22.3% patients were lost to follow up (Table IV).

In our study a total of 8 patients (10.9%) developed hypothyroidism between 6 months and 1 year of post radioiodine therapy. All of these patients had Grave's disease.

Discussion:

Thyroid disorders are quite common in Kashmir. Thyrotoxicosis is one of these disorders which demands prompt and permanent cure. Most of these patients are managed by antithyroid drugs and afew subjected to surgery. Since antithyroid drugsrarely induce remission in toxic nodular goitre and long term remission rate for Grave's disease is low [5,12], the patients need definitive therapy with I-131 or surgery. Radioiodine is increasingly considered the treatment of choice because of it's safety, ease of administration and high effectiveness in the management of thyrotoxicosis than by antithyroid drugs or surgery [11,13-15]. The most common complication of I-131 therapy is hypothyroidism which may develop many years after patient has been rendered euthyroid by I-131 or surgery [16], and if it does occur hormone replacement therapy is the only treatment of choice.

In our study the patients with solitary toxic nodule and Grave's disease showed 100% response to radioiodine, though 12.5% and 25.8% patients respectively needed a second dose of I-131 (Table IV). The least to respond were patients with toxic multinodular goitre where 25.9% of patients remained persistently toxic even after second dose of I-131. These results are consistent with the finding that toxic multinodular goitres require larger doses of I-131 for ablation [12]. Eight (10.9%) of our patients developed hypothyroidism within one year of treatment which is alow incidence as compared to results of other studies

[17]. Due to short period of follow up it will be premature to draw definite conclusions, because patients may develop hypothyroidism many years after I-131 therapy [16]. Hypothyroidism was observed only in patients of Grave's disease. The low incidence of hypothyroidism in toxic nodular goiter is explained by the fact that only the autonomous nodule concentrates radioiodine because rest of the gland is suppressed. This suppressed tissue resumes normal function after toxic nodule is ablated [6]. Most of our patients are still under follow up and some of the observations made above may alter if a prolonged follow up with a larger sample size is possible and if those patients lost to follow up return.

Most of our patients are living below poverty line and can not afford to buy the drugs for long-term therapy. Some of the patients belong to areas which remain cut-off for most of the wintermonths, thus there is no constant and reliable source of these drugs. Low level of education and poor motivation make drug compliance very difficult. In such a set up the option available for a patient is surgery or I-131. A substantial number of patients are elderly with associated medical problems making them unfit for surgery. Limited access to tertiary care hospitals dearth of hospital beds, inadequate expertise in the field of thyroid surgery keeps these patients in perpetual misery.

After a lapse of 3 years due to technical reasons, the department of Nuclear Medicine, S.K. Institute of Medical Sciences, Srinagar,

Table-I. Age and sex distribution of patients and type of hyperthyroidism

Type	*Below 25 yrs.*		*25-40 Yrs.*		*40-55 Yrs.*		*55-70 Yrs.*		*Total*
	M	*F*	*M*	*F*	*M*	*F*	*M*	*F*	
Diffuse	-	-	3	10	6	08	1	3	31
Solitary Nodule	-	-	0	07	0	06	0	3	16
Nodular	-	-	0	04	3	15	2	3	27
Total	-	-	3	21	9	29	3	9	74

Table-II. Values of T3 & T4 prior to I-131 therapy

Type	*T3 (ng/ml)*		*T4 (mg/dl)*	
	Mean	*Range*	*Mean*	*Range*
Diffuse	3.25	2.74-4.80	15.38	14.00-20.01
Solitary Nodule	4.10	2.50-4.84	17.41	14.68-20.30
Nodular	4.00	2.60-9.17	15.18	13.02-20.00

Normal Values at SKIMS: T3 = 0.7- 2.5 ng/ml.
T4 = 5.5-13.5 mg/dl.

Table-III. I-131 uptake values in various types of thyrotoxicosis

Type	At 2 hours (%)		At 48 hours (%)	
	Mean	Range	Mean	Range
Diffuse	29	09-57	50	29-62
Solitary Nodule	30	14-43	52	28-60
Nodular	28	13-50	52	28-60

Normal Values at SKIMS: At 2 hours = 3.26 ± 1.6% (Mean±2SD)
At 24 hours = 13.2 ± 4.9% (Mean±2SD)

Table-IV. Response rate to I-131 therapy

Type	*Ist. dose No.of pts.*	*%age*	*2nd dose No. of pts.*	*%age*	*Total %age*
Diffuse	23/31	74.2	8/31	25.8	100
Solitary Nodular	14/16	87.5	2/16	12.5	100
Nodular	08/27	29.6	6/27	22.2	51.9
Total:	45/74	60.8	16/74	21.6	82.4

Table-V. Incidence of hypothyroidism after I-131 therapy

Type	At 6 months		At 1 year		Total
	No.of Pts.	%age	No.of Pts.	%age	(%)
Diffuse	3/31	9.7	5/31	16.1	25.8
Solitary Nodular	0/16	0	0/16	0	0
Nodular	0/27	0	0/27	0	0
Total	3/74	4.1	5/74	6.8	10.9

has started its thyroid clinic. Since 1993, the patients of thyrotoxicosis are undergoing diagnostic workup and radioiodine therapy. The objective of this study is follow up of patients after I-131 therapy and also to impress upon practitioners that an effective, safe and cheap modality of treatment for thyrotoxicosis in the form of radioiodine is available in this part of the world.

Correspondence to:
Dr. Syed Mushtaq,
Consultant Department
of Nuclear Medicine,
Institute of Medical Sciences,
Soura, Post Bag NO: 27,
GPO Srinagar.

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News: Dr. Gh. Jeelani Mufti (FRCP) has been promoted as Professor of Medical Oncology at KING'S College of Medicine and Dentistry London this year. The editorial board and advisory commitee members congratulate him. He is the 1st Kashmiri doctor (graduated from Medical College Srinagar in 1972,) to be Professor in this prestigious institution in U.K. Dr. Mufti is also consultant editor of JK-Practitioner and contributes a lot in its progress.

Original Articles:

"Effect of Fluoride ions on the thyroid glands of Guinea Pigs"

***Mahmood, **G.H. Bhat**

Abstract:

The present study was conducted to evaluate the effect of sodium fluoride on the thyroid glands of guinea pigs. Microscopy of the thyroid glands with low concentration of the sodium fluoride showed vacuolations in the colloid with increased concentration there was:

1. Depletion of colloid from the follicles.
2. Shrinkage of follicles.
3. Disruption of follicular basement membrane associated with oedema and degeneration of the follicular epithelial cells.
4. Increased follicular vascularity.
5. Fatty degeneration in the inter-follicular connective tissue.

JK Practitioner 1996; 3:2 94-96

Introduction:

Ogilive 1953 (I) observed in thyroid a reduction in size of the nuclei, reduction in the amount of the cytoplasm in the cells and decrease in the cell medium. The follicular cells however remained columnar and were clearly approximated to each other. There was an increase in inter-lobular and intra-lobular connective tissue.

Grabski 1972 (2) after four weeks with fluoride 1.0 mg per day showed a sufficient increase in the relative and absolute thyroid weight associated to an increase in the connective tissue. There was decrease in the area occupied by follicles in comparison to the control.

Material and Method:

A total of 100 guinea pigs were taken and divided into four groups 25 each. Group 'A' was given drinking water containing 10 ppm of sodium fluoride in one litre of water. The group 'B' received 500 ppm of sodium fluoride in one litre of water. The group 'C' was given 1000 ppm of sodium fluoride in one litre of water. The group 'D' served as control. The group A,B,C and D were fed on the standard diet. The animals were observed for changes in the gross appearance and body weight. The experimental animals and control group were killed after 30, 60 and 90 days. The thyroid specimens obtained were processed for histopathological examination. Staining was performed by Haematoxylin and Eosin stain.

Results:

The histological examination of the organs were categorised as A, B and C groups. The groups A, B, and C were further subdivided into A_1, B_1, C_1(After 1st month), A_2, B_2, C_2 (After 2nd month)and A_3, B_3, C_3 (After 3rd month of the fluoride treatment) respectively.

Group A

The thyroid gland of A and B group showed some vacuolations of the colloid material of the follicular cells. The group A_3 showed obvious changes in the size and pattern of the follicles. The follicles were found smaller in size with disruption of the follicular epithelium at places. The epithelial cells of the follicles were found oedematous. The colloid material was found depleted from some of the follicles. The connective tissue and vascularity was found increased —— Fig (1).

Group B

Group B_1 showed shrinkage of the follicles. The epithelial cells of the follicles were oedematous. There was an increase in the inter-follicular connective tissue and vascularity.

In group B_2 there was further shrinkage in the size of the follicles. The follicles showed vacuolations in their colloid content. The inter-follicular connective tissue and vascularity was more increased.

In group B_3 the follicles showed shrinkage of size. Most of the follicles were empty. Depletion of the colloid material of follicles was more in this group. Some of the follicles were broken. Patches of degenerative follicles were also seen occassionally. There was an increased inter-follicular connective tissue and vascularity.

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* Lecturer

** Prof & Head Deptt. of Anatomy,
Govt. Medical College Srinagar.

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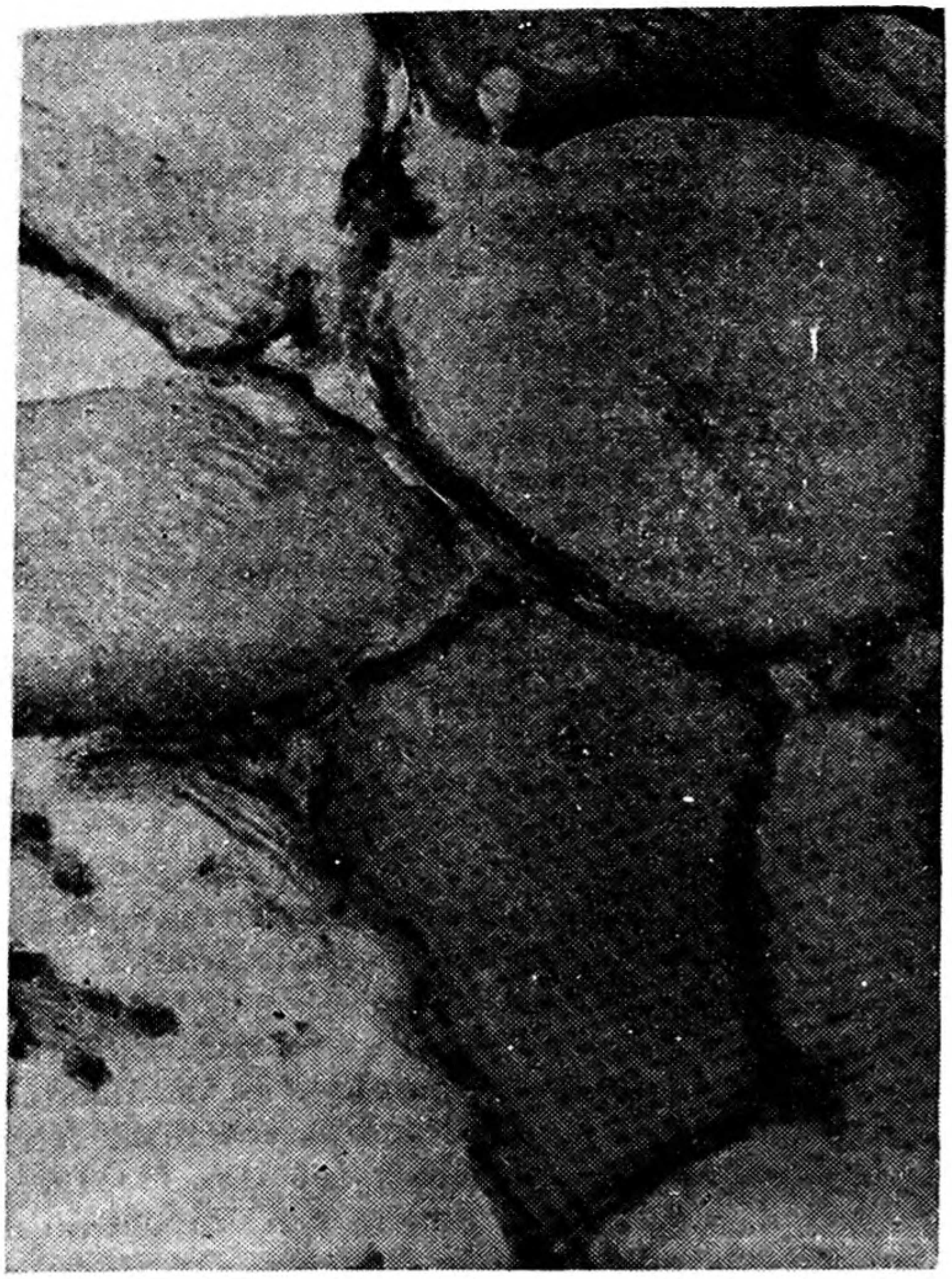

Fig. 1

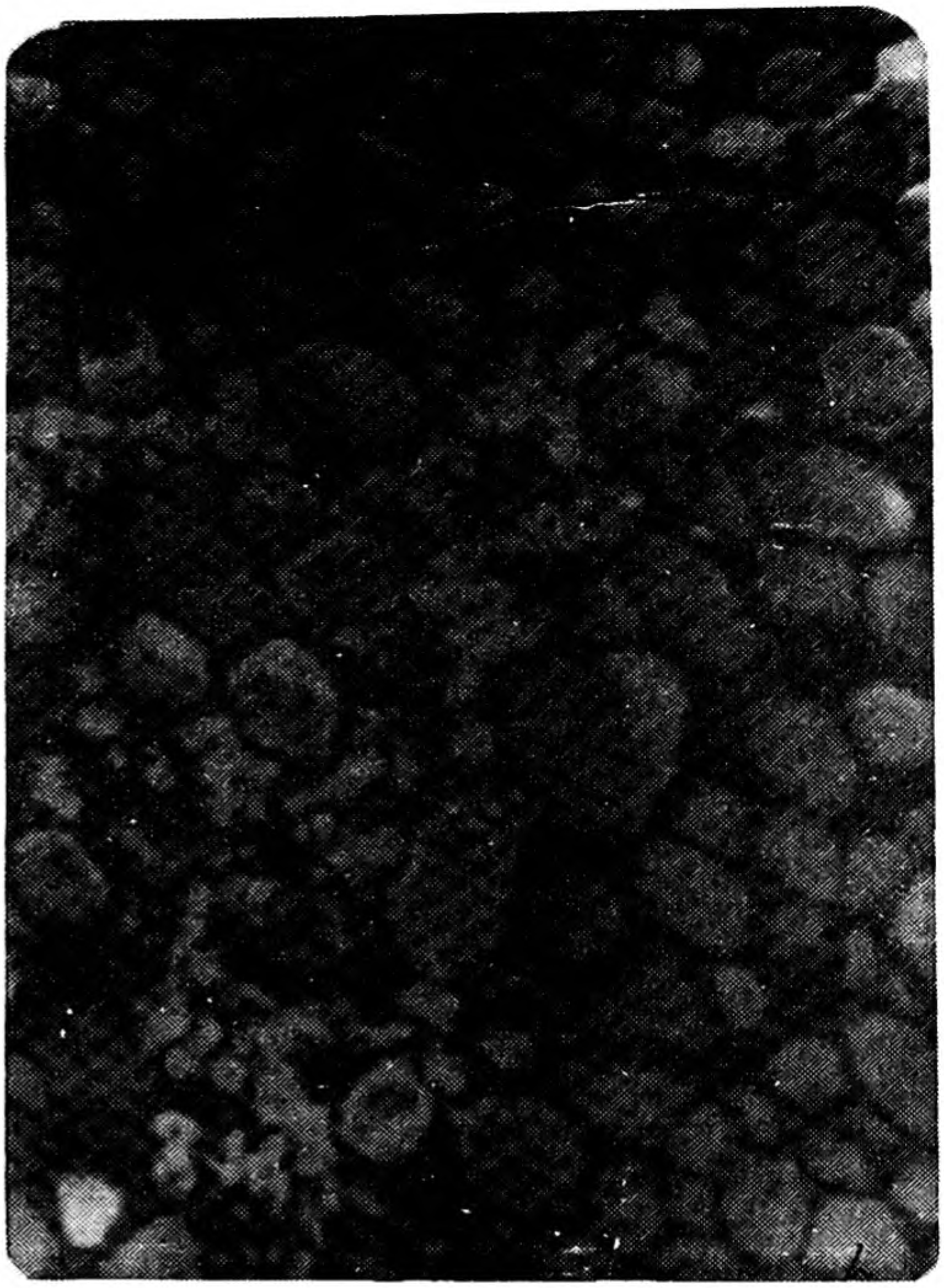

Fig. 2

Group C

In Group C_1 the follicular size was reduced. The colloid material within the follicles had almost disappeared. Most of the follicular epithelial cells showed disruption and degeneration. The basement membrane was seen disrupted. Some fatty infilteration was observed in inter-follicular tissue. The vascularity was much more increased. In group C_2 size of the follicles was reduced with disruption of the basement membrane. There was no colloid material in the follicles. The epithelial cells of the follicles showed degenerative changes. There was more fatty infiltration of the inter-follicular tissue. The vascularity was slightly increased as compared to above groups —— Fig (2).

Group C_3 showed loss of normal architecture of thyroid. The colloid content of the follicles has disappeared completely. The epithelial cells of the follicles showed degeneration. There was marked fatty infiltration. The inter-follicular connective tissue was found highly increased.

Discussion:

The involvememt of the thyroid gland has received varied opinion from time to time. It was Blister et al (3) who observed, hypertrophy, hyperplasia and increased colloid with-drawal of the gland following fluoride.

Ogilive (1) reported that microscopic changes in thyroid glands of fluoride treated animals were indicative of atrophy associated with reduction in the size of nuclei and cytoplasm.

Demol (4) failed to detect any specific change in relation to thyroid function tests after excessive fluoride administration. In 240 patients with normal thyroid function, Kick et al 1953 (5) and Roholm 1937 (6) could not elucidate any change in thyroid following fluoride administration.

In the present study with strength of 500 ppm the follicles showed a shrinkage in size which was later associated with disruption of the basement membrane. These findings are similar to the observation made by Ogilive (1). The intercellular connective tissue was at its maximum at 1000 ppm strength. With the strength of 10 ppm the colloid exhibited small vacuolation. The follicles appeared empty with higher concentration of sodium fluoride. The increased vascularity, and fatty degeneration of epithelium was observed in animals fed with a strength of 1000 ppm of sodium fluoride. Such findings have not been reported by earlier workers. Philips, et al 1933 (7), Chang, et al 1934 (8) indicated that fluoride is stored to a great extent by the thyroid as compared to other organs. This fact supports our findings that due to the cummulative deposition of fluoride the thyroid is affected to the maximum and changes are more pronounced with higher strength. Roholim 1937 (6) and latter Hodge and Smith 1965 (9) and Day et al 1972 (10) produced goitre in experimental animals after the administration of sodium fluoride. The coincidence of dental fluorosis and goitre was reported by Wilson 1941 (11) in England from area of Somerset. Michael et al 1967 (12) detected goitre in 27.4% of human individuals suffering from fluorosis. The size of the thyroid glands was five times more than that of the normal. The biochemical investigation conducted by Siddique 1960 (13) did not substantiate this view. In close confirmity with the observations of Siddique (13) Jolly et al (14) did not find evidence of goitre in 26 cases of endemic fluorosis. Goldembrg 1938 cited by Green Wood 1940 (15) admitted this beneficial effects of fluoride in toxic goitre to an in-vivo inactivation of thyroxine and inhibition of oxidoses and other tissue ferments.

In view of known relationship between thyroid and hallogens, it is logical that fluoride ions are stored in thyroid glands and would cause deliterous effects as degeneration and fatty infiltration.

The above observations also suggest that fluoride compounds in therapeutic doses could be used as anti-thyroid drugs in the treatment of hyperthyroidism. However, this suggestion needs further investigations, particularly at bio-chemical level.

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Case Reports:

"Massive Splenomegaly, an Unusal Presentation of Echinococosis"

***Shiekh Aejaz Aziz, Mushtaq Ahmad, Mohammad Amin Akhter,
Gul Mohammad Bhat, Ajaz Lone, Ghulam Nabi, Hajra Bano**

Abstract:

A 25 years young, married female, presenting with progressive abdominal distention and secondary amenorrhoea, was confirmed to have an extensive Hydatidosis. She has been prescribed Albendazole 20 mg/kg body weight/day PC, initially for a period of eight weeks.

JK Practitioner 1996; 3:2 97-98

Key words: Echinococosis, Massive splenomegaly.

Introduction:

Echinococosis (Hydatid disease) is a zoonosis, caused by larval cestodes in man, acting as imtermediate host. The lesser extent of splenic involvement in Hydatid disease is known (I), however, a massive splenomegaly with Hydatid disease is unusual.

Case Report:

Ms MK, Gujjar lady, was admitted in the Department of Medical Oncology, Institute of Medical Sciences, Srinagar on 08.04.1995, with complaints of progressive distension of abdomen, secondary amenorrhoea and intermittant low grade fever, of one year's duration. Clinical examination revealed her in malnourished state with severe anemia, dependant oedema having no stigmata of chronic liver disease peripherally. Per-abdominal examination revealed splenomegaly, 19cms below costal margin (left), having smooth surface with a visible notch, Hepatomegaly (mainly left lobe) 5cms below right costal margin, and also two glubular swellings in epigastrium and hypogastrium, measuring 4x4 cms and 2.5x4 cms respectively. There was no ascites. Examination of the chest revealed features of minimal pleural effusion, posteriorly on the right side. On P/V examination, uterus size could not be made out and there was vaginal stenosis. Rest of the clinical examination was non-contributory. Possibilities of a myeloproliferative disorder, Hairy cell leukemia were thought. Adult onset storage disease and Hydatidosis were kept in mind. The investigations showed Hb 7 gm%, TLC 7000/mm3, DLC-P62, LI8, E20, ESR 06 mm/hr, PBF showing hypochromic microcytic anemia. Urine and sputum examination were normal. LFT showed serum bilirubin 0.4 mg%, total proteins 5.Ig%, albumin 2.6 g%, alkaline phosphatase 554 Y/I (Normal values upto 300 U/I), acid phosphatase 2.9 U/I (Normal 4 U/I), calcium 7.9 mg%. Coagulation profile revealed PT 18 seconds (control I2 seconds), PTI 66.6% PTTK 36 seconds (control 30 seconds). Chest X-ray revealed a glubular mass occupying more than 2/3rd of right hemithorax, with oedoma of the horizontal fissure, pushed upto first interspace **(Figure I)**. Skeletal survery was normal. Abdominal USG revealed multiple trans-sonic areas with well formed walls, smooth in outline having no internal echoes, septations or calcifications with acoustic shadowing, in liver, spleen, kidneys, and in relation ot mesentry and ovaries **(Figure 2)**. Casoni's test was positive (immediate). Hydatid serology was strongly positive with titres I; I280 (ELISA). She was labelled to have a disseminated echinococcosis, and put on Albendazole, initially for 8 weeks, per orally.

Discussion:

Echinococosis is encountered worldwide with pronounced epidemiological and economic importance in cattle and sheep raising areas. The patient under discussion is a Gujjar, which essentially is a nomadic community, having an intense assosiation with cattle and domestic herbivorous animals. Hydatid disease exists in Hyperendemic (Sub-saharan grasslands of Africa_, endemic (Australia, New-zealand) and in extinction states (Iceland) [2].

Hydatid disease is caused by the laryal stage in man acting as intermediate host of the cannine Tapeworm, Echinococus granulosus, which normally has its intermediate stage in sheep or other herbivorous animal (unilocular Hydatid) and Echninococus multilocularis having its normal intermediate stage in rodents. The incubation period rages from 10-30 years, except in Brain and ets, where cysts are present in childhood [3]. More than 75% patients present with abdominal distension, with liver, especially right libe, as commonest site (60-90%). Other less common involved sites include lungs (30%), kidney, heart, spleen, subcutaneous tissues, bones and brain [4]. The clinical features

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*Department/s of Medical Oncology and Internal Medicine,
Sheri-Kashmir Institute of Medical Sciences,
Srinagar, India.

Fig. 1 X-ray Chest.

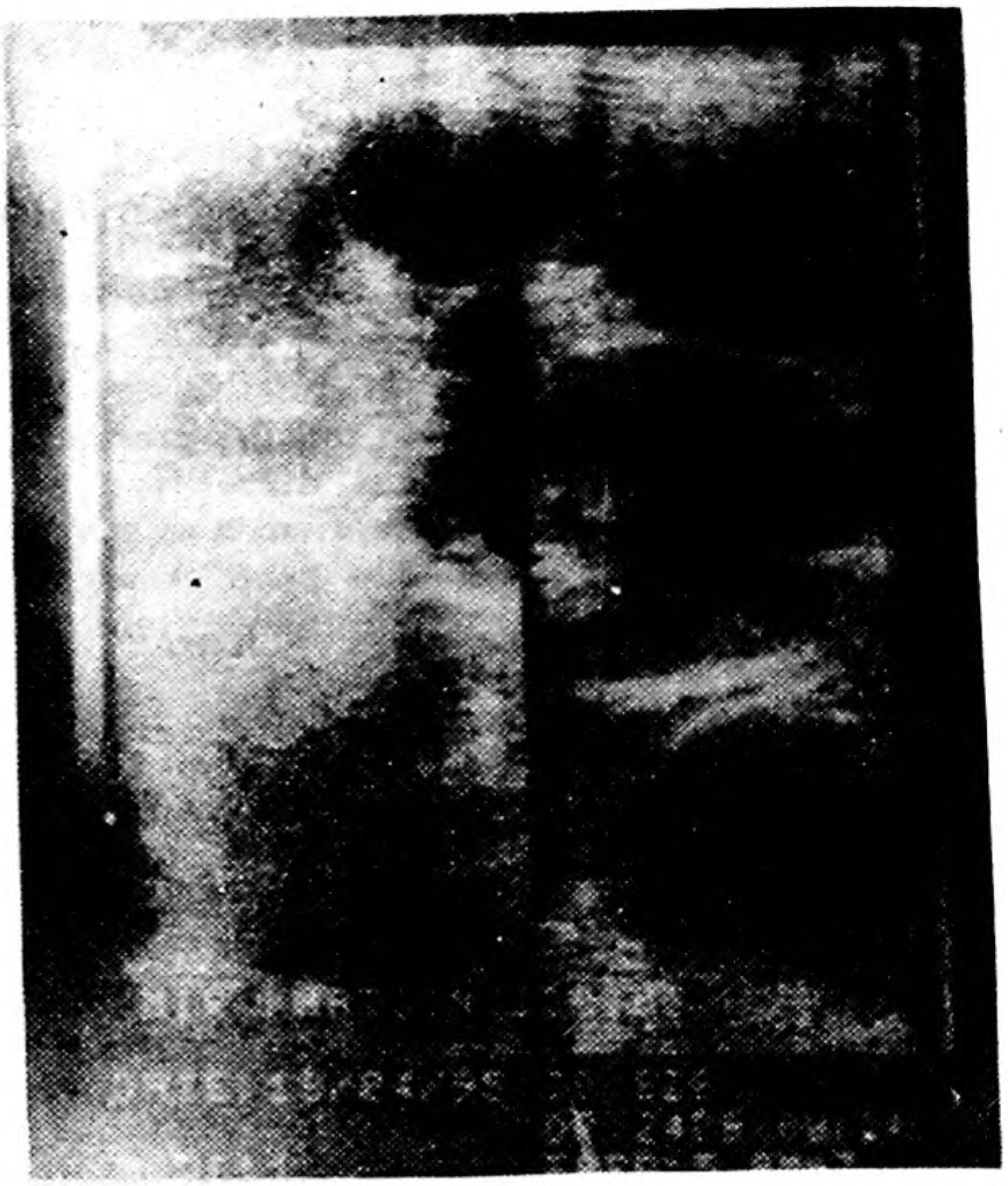

Fig. 2 Ultrasound Abdomen.

are variable and depend on the space occupation of the growing eyts, growing at the rate of I cm/year [5], rupture (cholangitis, peritoneal spread) or rarely due to the allergic symptoms. Unusual features include lung collapse, dysphagia, Horner's syndrome, Superior vena caval compression, Portal Hypertension, myopathy, space occupying lesion in ventricles of heart and hematuria [3]. A massive splenic enlargement indicating splenic extension to umblicus and beyond and Hydatid disease does not figure a common cause of the same, as was seen in the patient under review. These Hydatid cysts on an average are 0.5-IL in size, however, the size may be as mammoth as to occupy 57 liters of fluid. About 14% cysts resolve spontaneously [1].

Control and prevention of the infection depends upon breaking the chain of transfer from sheep/ cattle to dogs (deworming) and good house hold and personal hygiene. The treatment is surgical (decompression, enucleation), medical (Mebendazole, Albendazole, Praziquental) or combined (prevention of recurrence, spillage). To our knowledge, we are yet to find a similar presentation in literature, so the follow-up of the patient under discussion and a caution, to include Echinococcosis among the causes of massive splenomegaly, though rare, should be kept in mind.

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***For Correspondance:**
Dr. Sheikh Aejaz Aziz, MD, DM.
Consultant, Department
of Medical Oncology,
SK Institute of Medical Sciences,
Post Box No: 27,
Soura, Srinagar-190011, India

Case Reports:

Adenoma Upper Lip (Papillary - Type)

*Bashir Ahmed, **M. Shafi, **Nowshaba Rashid

Abstract:

Papillary adenoma upper lip is one of the rare tumours in upper labial vestibule. The article deals with the case of such a lesion found in upper lip of a 50 year old female from a village. The significance of the lesion is recurrence and continious disfigurement of the face, although the lasion seems to be benign in nature. *JK Practitioner 1996; 3:2 99*

Introduction:

Papillary Adenoma is a minor-salivary gland tumour which occurs in the oral cavity and is a rare anamoly involving upper lip. It generally presents as a slowly growing well circumscribed, firm, nodule particularly in upper lip which is not fixed and may be moved a little through tissues.

Case Report:

A 50 year old female from village presented to the department of Oral Surgery of Govt. Dental College, Srinagar, with the complaint of swelling over the upper lip for the last six months. The lession as per history was previously intervened by some surgeons, and recurred after six months. Patient was worried about her aesthetics as the facial look seemed obliterated. At the time of examination, the lesion, located in upper lip was about 1 cm. X 3 cm. in diameter, hard in consistency and non tender.

X-Ray Skull (A.P. view and I.o.P.A view) revealed an interesting radiolucent lesion extending upto the left nasal fossa indicating its perforation. (Fig-I) However, clinically there was no such perforation.

Treatment:

On preliminary histopathological report it was decided to enucleate the mass for further comfirmatory tests. The whole mass was circumcised and preserved for biopsy which latter confirmed as papillary type adenoma.

After hospitalisation, the patient responded well and improved by way of aesthetics, speech and the healing was uneventful. Patient was seen after 3 months and was found absoutly with no sign of recurrence and was advised to go for prosthodontic replacement of lost anterior teeth, to add up in her aesthetics and speech.

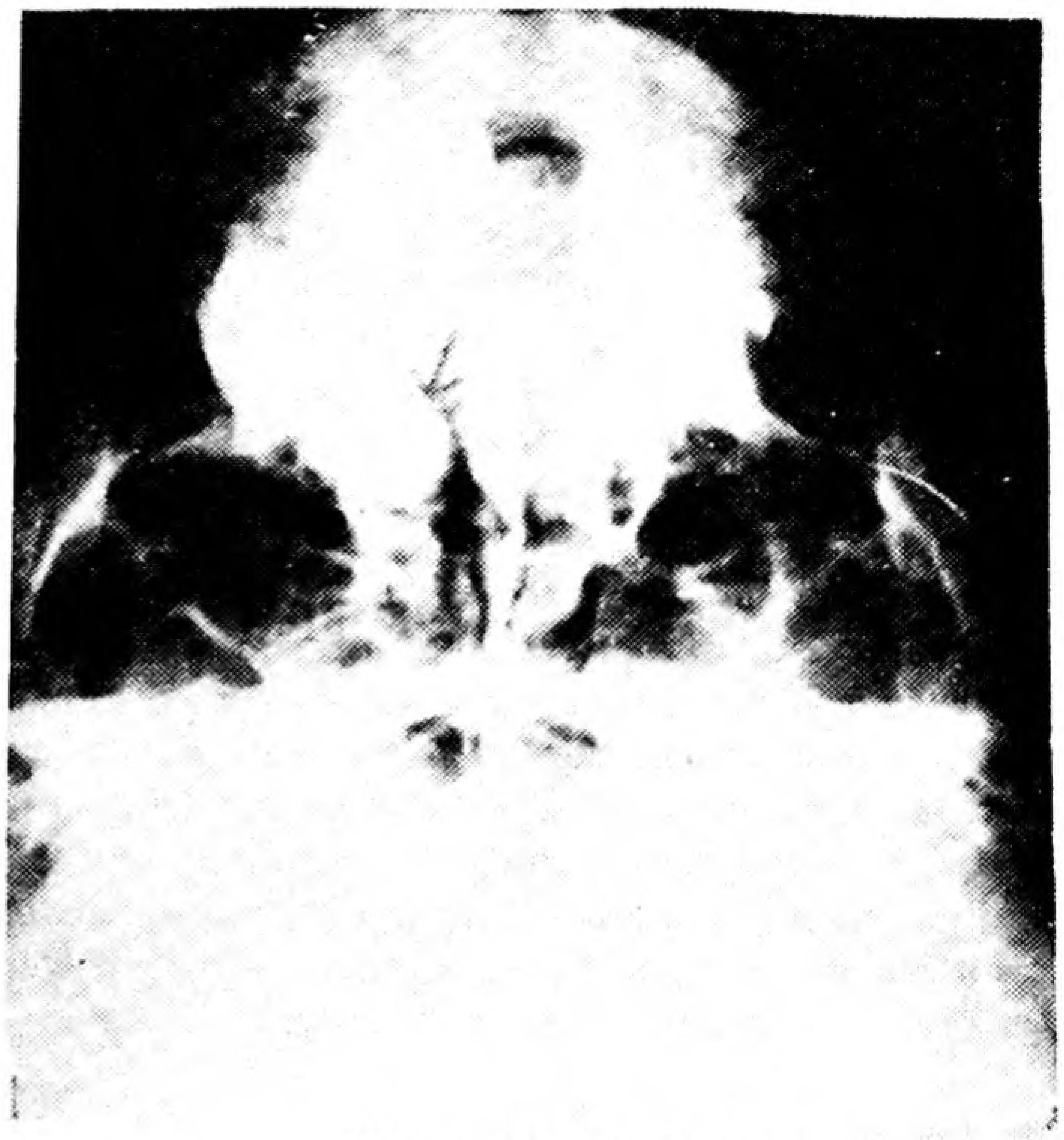

Fig. 1 X-ray Skull.

Discussion:

Papillary adenoma is an exophytic, elevated, solitary, sessile nodular firm swelling (growth) present on upper lip. The lesion as found clinically could give the impression of some other entity like fibroma, angioedema, lipoma, mucous retentioncyst or nasolabialcyst. However histopathological and clinical assessment excluded all other entities and confirmed thediagnosis of papillary type adenoma which has been attributed to some morpho-logical changes in the luminal-cells of minor salivary gland duct.

In this particular case the history has added to the confirmation of diagnosis of papillary type adenoma as this lesion has also recurred within a period of six months. One should make good attempt to excise the lesion meticulously so as to minimize the chances of recurrence. This particular patient was followed for a pretty long time and when last seen no recurrence was detected.

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* B-Grade Specialist
** Resident Deptt. of Oral Surgery
Govt. Dental College, Srinagar, Kashmir.

Case Reports:

" Body Favus - A Case Report "

***Qazi Masood, **Sheikh Manzoor**

Abstract:

Body favus is a rare condition in which glaborous skin is infected by trichophyton schoenleinii. Here we report a case of body favus.

JK Practitioner 1996; 3:2 100

Case Report:

A 15 year old female from rural area reported to us, with a history of yellowish patches on her body for last one year.

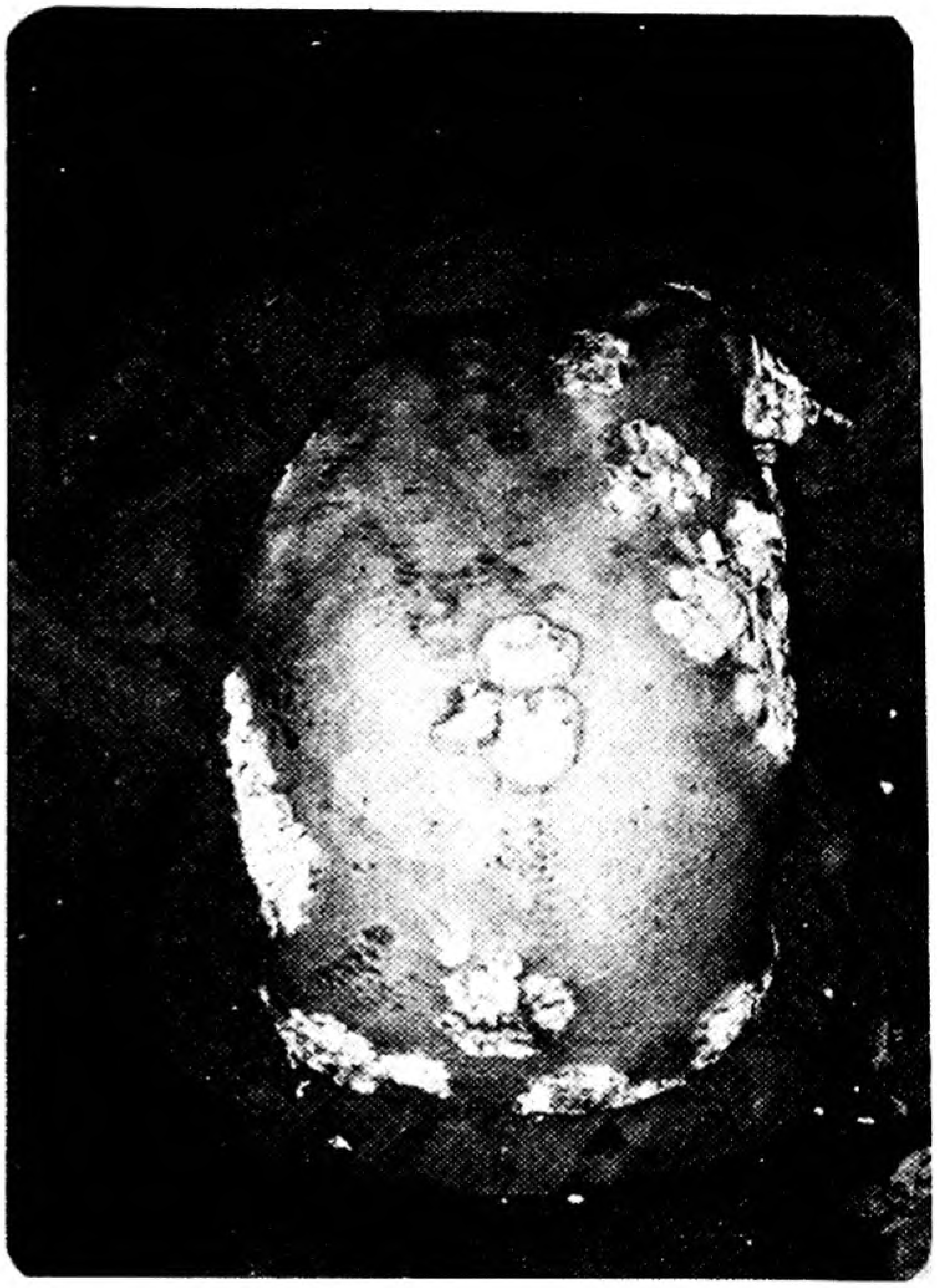
Her physical examination revealed only conjuctival pallor and decreased nutritional status. Her hair, nails and mucous membranes were normal. Her systemic examination was normal. her body showed multiple extensive yellow crusts (Fig. 1). Her routine investigations were normal except anaemia. Microscopic examination of the skin smear showed the presence of fungus. The culture of the smear revealed trichophyton schoenleinii fungus.

Discussion:

Favus of scalp is not a rare entity. It is seen sporadically in a variety of countries from South Africa[1] the middle East Pakistan, USA and the UK to Australia.

In Kashmir it is relatively common.

The classical pictures of T. capitus due to the organism is characterized by the presence of yellowish cup-shaped crusts known as scatula[2]. Each scutulun develops round a hair which pierces it centrally. Adjacent crusts enlarge to become confluent and form a mass of yellow crusting. Many patients, may show less distinctive changes in early cases perhaps amounting to no more than perifollicular redness and some matting of the hair. Extensive patchy hair loss with cicatrical alopecia and atrophy among patches of normal hair may be found in long standing cases where much of the hair loss is irreversible. In such patients the glabrous skin is commonly affected by the development of similar yellowish crusts[3]. In our cases there was no evidence of scalp involvement. Only glabous skin was involved. About 2-3% have nail involvement[4] but in our case nail were normal.



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* Asst. Professor
** Resident
Deptt. of Dermatology
Govt. Medical College Srinagar

Case Reports:

"Mexiletine Toxicity - A Case Report"

*** S. B. Banzal, ** H. S. Rissam, * F.A. Bhat, *** J. Ashraf**

Abstract:

We describe a case of Mexiletine poisoning, consumed for suicidal purpose. Patient developed neurological toxic manifestations without much cardiac problem. Mexiletine poisoning is rarely reported in literature. This is the second report in literature with such a high dose to our knowledge.

Mexiletine is being commonly used for its antiarrhythmic action by oral and IV routes during last ten years[1]. Toxicity due to overdose of antiarrhythmics is described in literature but Mexiletine poisoning has not been often reported, because of well-monitored administration of this drug and because suicidal tendencies are not common in cardiac patients as compared to what is seen in psychiatric population.

JK Practitioner 1996; 3:2 101-102

Case Report:

We present a case who allegedly consumed 4gm of Mexiletine capsules from one family member's medicine-kit for suicidal reasons. This twenty year old Saudi male was referred to King Fahd Central Hospital, Gizan, with history of "accidently" ingesting 20 capsules of Mexiletine (200mg each). Within two hours he felt dizziness and became drowsy. He was taken to nearby general hospital, where he was detected to be in semi-comatose state with no localizing sign. Cardiovascular system was normal. He was given a gastric lavage there, and was referred to us for further management.

On arrival to our hospital he was drowsy but moving all limbs. Pulse 70/mt, regular, BP 139/90 mmHg, other systemic review was normal, R/R 18/ minute.

After sometime, patient developed coarse tremors of hands. He was given I.V 5mg of Diazepam following which he settled. BP dropped to 100/50 mmHg, heart rate remained between 70-80/minute. There was no tachycardia and no arrhythmia. Basal ECG and sequential ECG's were normal.

After about 12 hours he developed gross truncal ataxia and horizontal nystagmus. No other involuntary movement or any other neurological deficit was noticed. Ataxia and nystagmus disappeared within 24 hours. Patients became fully alert and conscious. His cardiovascular system remained stable. He was discharged after three days of observation without any residual problem.

Discussion:

Mexiletine is a class 1-B antiarrhythmic agent which depresses the maximum rate of depolarisation with little or no modification of resting potentials or the duration of action potentials[2].

It is commonly used drug for treatment of ventricular arrhythmias [3,4]. Side effects of Mexiletine are mainly related to plasma concentration and depend on individual reaction to plasma levels. During treatment with Mexiletine frequent side effects seen are indigestion, hiccups, unpleasant taste nausea, vomiting and arthralgia. Neurological side effects described are drowsiness, dizziness, confusion, light headedness, dysarthria, blurred vision, nystagmus, tremors, paraesthesias, ataxia and convulsions. Reported caddiovascular problems are sinus bradycardia, hypotension, atrial fibrillation, A-V dissociation, and palpitations with or without ventricular ectopic activity. Rare reports of liver dysfunction, skin rashes have been described[1].

Its elimination half life is 10 hours[5]. Mexiletine has a narrow therapeutic ratio and many adverse effects are dose related.

Our patient had ingested 4 grams of Mexiletine. He showed neurological side effects like tremors, truncal ataxia, nystagmus, confusion and drowsiness. He developed transient fall of BP (40mm Hg) from the baseline level, but developed no electrocardiographic abnormality.

The case is being reported for the general interest of its rarity. In review of literature authors have come across only one case report[6] of a young man, aged 22 years, who consumed almost similar

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* Medical Specialist, ** Senior Consultant cardiologist, *** Chief of Internal Medicine Deptt. Deptt. of Medicine, King Fahd Central Hospital, Gizan, PO Box 204, GIZAN, Kingdom of Saudi Arabia.

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dose (4.4 grams) of Mexiletine. He developed serious side effects including convulsions, cyanosis, unrecordable blood pressure, complete heart block, asystole and finally death despite all supportive measures.

Our case has shown comparatively a very benign course, possible because of early hospitalization, timely gastric lavage, underlying normal cardiac status and his young age.

Acknowledgement:

The Secretarial assistance of Mr. Moncy Varghese is greatly acknowledged.

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"Tuberculous Parotitis"

***N. A. Bhat**

Abstract:

Tuberculosis of the parotid gland is rare. It cannot be distinguished from a parotid tumor on clinical examination so surgical exploration has usually been required for diagnosis. We present a case with the same diagnostic dilemma.

JK Practitioner 1996; 3:2 102-103

Keywords: Parotid gland; Tuberculous Parotitis; Superficial Parotidectomy.

Introduction:

Recent years have seen a decline in the incidence of tuberculosis in Britian, although it remains relatively common in Asian population [1].

Diffuse, infective parotitis is common, usually resulting from the mumps, echo or Coxsackie A virus or staphylococcus in the elderly and debilitated. Tuberculosis in the parotid region however is very uncommon, even in countries where the disease is otherwise rife. It nearly always presents as a localised mass, resulting from infection of intra-capsular or periglandular lymph nodes. Diagnosis is therefore often made after operative excision of the presenting mass.

Parenchymatous tuberculosis affecting the entire gland is extremely rare. Only two cases are recorded in the salivary gland register of Germany for the period 1965-1981[2] and one further case in the English literature in the last 10 years[3] (Stanley et al 1983). In the patient reported here the condition was unassociated with a primary pulmonary focus.

Case Report:

A 57 year old Asian gentleman, resident in UK for the last 20 years, presented with a swelling of the left parotid region of 10 years durations. The swelling had been increasing in size for 4 months. There was no history of pain, weight loss, night sweats or chest symptoms. Examination demonstrated a 1x1.5 cm, mobile, firm, non tender swelling in the lower hald of left parotid gland. There was no facial nerve weakness. Examination of neck, mouth and pharynx was normal.

Investigation revealed a white cell count of 4.41x109/L and lymphyte count of 1.19x109/lt. X-ray of chest was normal. Our initial diagnosis was pleomorphic adenoma and left parotid region was explored with a view of doing a superficial parotidectomy. Exploration revealed a tumour mass extending into the deep lobe with no clear distinction between the tumour and the normal parotid gland and Facial Nerve was going into the mass. Frozen section was done which gave a probable diagnosis of sarcoidosis. A biopsy specimen was taken and the operation was terminated.

Histology Report:

Macro-Report:"Salivary gland 5 x 2.5 x 2.3 cms serial slicing shows fleshy infiltration and a small lymph node 1 cm in size"

Micro-Report:"Both lymph node and salivary glands are almost completely replaced by typical sarcoid granulamata. However, one can never be sure and after a long search one A F B (acid fast

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* Registrar E. N. T. Deptt.
10 - Torrant Walk
Walsqrave Conventry (U.K.)

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bacillus) was found, confirmed i.e. this is truberculosis parotitis; patient was referred to the physicians for further management.

Discussion:

Mycobacterial parotitis is very uncommon. It was first described in 1894 by Von Stubenrauch[4]. Since then only 43 cases have been reported in the English literature and of these the majority were seen before 1941 [5]. Two pathological types occur, a common focal form resulting from lymph node infection and a rare diffuse form resulting from parenchymatous involvement. Of the 13 reported cases in the last 10 years, 12 are of the focal form[1,3,4,6,7] and only one of the diffuse form [3]. Before pasturization mycobacterium bovis was the commonest organism but now it is the mycobacterium tuberculosis. In the lacalized form, resulting from infection of intra or peri glandular lymph nodes, the pathogenesis is probably similar focus. However, the origin may have arisen from an initial site in the tonsil (Wilmot et al 1957). The patient is often systemically well and the chest X-ray may be normal. In 75% of cases there is no family or personal history of tuberculosis[8]. The mass may frequently be mistaken for a parotid neoplasm - as in our case resulting in fine needle aspiration or surgical excision with the risk of subsequent fistula formation[3].

The second form, with diffuse involvement of the gland parenchyma, is extremely rare[3,8]. In Stanley's case the patient developed a parotid abscess and histological examination revealed ceseating granulomas of the peri-glandular nodes as well as the parenchyma. There was no evidence of active disease elsewhere and so it is probable that this case was of primary nodule disease with secondary spread to the surrounding gland. This is likely mechanism in our case.

The diagnosis is difficult to make given its extreme rarity and similar clinical picture to other forms of inflammatory parotitis. In appropriate surgical exploration and delay in instituting correct treatment may result in gland destruction and fistula formation or possible cross infection from an open pulmonary focus.

Conclusion:

Tuberculosis of the parotid gland is rare. The diagnosis is difficult to make clinically and even at operation the tuberculosis lesion is usually considered to be a tumour. In our view, the tuberculus parotitis should be considered a strong possibility in the Asian population who present with a long standing asymptomatic parotid lumps. Fine needle aspiration can be used safely for diagnosis purposes.

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Special Article

Islamic View on Organ Transplantation

*M. A. Albar
FRCP

Abstract:

This parer discusses the ethical and Islamic Jurisprudence view on the subject of organ transplantation, which is expanding very rapidly to involve many Islamic countries. The Kingdom of Saudi Arabia is playing a major role, both in formulating the Islamic jurisprudence views and implementing them on its proliferating centres for organ transplantation (5 centres for kidney transplant, 1-2 for cardiac transplants and proposed centre for liver transplants).

The historical background of the Islamic jurists views on organ transplantation are discussed briefly. Islamic jurists sanctioned bone and teeth transplantation since one millennium. The recent Fatwas and various decrees of Islamic jurists conferences were discussed, and the underlying Islamic rules regarding transplantation were highlighted. More and more organs and tissues are transplanted with the resulting increased need for donors, who could be living or cadaveric.

The third International conference of Islamic jurists held in Amman 1986 accepted the concept of Brain Death and equated it with cardiac death. This paved the way for the rapid expansion of organ transplants project especially in Saudi Arabia, the leading Islamic country in cadaveric organ transplants were 200 kidneys cadaveric transplants and 12 cardiac transplants were performed.

The 6th International conference of Islamic jurists held in Jeddah March 14-20, 1990 addressed new issues and frontiers of organ transplantation e.g. transplantation of nervous tissues, anencephalics, embryos and reproductive system.

This article discusses these Fatwas and decree and their implications.

JK Practitioner 1996; 3:2 104-108

Introduction

Islam is a religion that differs from many other religions. It encompasses the secular with the spiritual, the mundane with the celestial. It provides a code of life.

Man is the vicegerent of God (Allah) on earth. "Behold thy Lord said to the angels: I will create a vicegerent on earth"(1). "He fashioned man in due proportion and breathed into him something of His spirit"(2).

Not only was Adam honoured by Allah, but his progeny also, provided they trod on the right path. "We have honoured the progeny of Adam. provided them with transport on land and sea; given them for sustenance things good and pure; and conferred on them special favors above a great part of our creation."(3)

Human life begins at the time of ensoulment, which is stated in the sayings of the Prophet to be the 120th day from the moment of conception. (4) Prior to that moment the embryo has a sanctity, but not reaching that of a full human being.

The life ends with the departure of the soul (or spirit), a process which cannot be identified by mortals except by the accompanying signs; the most important of which is the cessation of respiration and circulation.

The sanctity of the human body, however, is not lessened by the departure of soul and declaration of death. The human body whether living or dead should be venerated likewise.

The Prophet Mohammed (PBUH) rebuked a man who broke a bone of a deceased which he found in a cemetery. The Prophet said "The sin of breaking the bone of a dead man is equal to the sin of breaking the bones of a living man." (5)(6)

The dead body should be prepared for burial as soon as possible, in order to avoid putrefaction which occurs rapidly in hot climates. Cremation is not allowed.

Due respect and reverence should be given to the funeral. The Prophet himself stood in veneration for passing by funeral of a jew, at a time when jews were bitter enemies. One of the companions of the

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*Consultant of Islamic Medicine, King Fahd Medical Research Center, King Abdulaziz University, Jeddah, Saudi Arabia.

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Prophet exclaimed: "It is the funeral of a jew", The Prophet answered. "Is it not a human soul?"(7)

Historical Background

Organ transplantation is not a novelty of the 20th century. Indeed it was known in one form or another even in prehistoric eras. Ancient Hindus surgeons described methods for repairing defects of the nose and ears using autograft from the neighboring skin, a technique which remains to the present day. Susruta Sanhita, an old Indian medical document written in 700 B.C. described elegantly the procedure which was emulated by the Italian Tagliacozzi of the 16th century and the British surgeons working in India late in the 17th and 18th centuries.(8)

Teeth transplantation was practiced in ancient Egypt, Greece, Rome, Pre-Columbian North and South America. The Arab surgeons were experts at this technique one thousand years ago.(9) (10)

At the time of Prophet Mohammed (PBUH) (570-632 AD) one of his companions called Qatada ibn Noorman lost his eye during the battle of Ohod. The Prophet replanted it back and it became best of his two eyes. (11)

In the battle of Badir the Prophet (PBUH) replaced the arm of Muawath bin Afra and the hand of Habib bin Yasaf. (12)(13)

Muslim jurists sanctioned transplantation of teeth and bones which was practised by Muslim surgeons since one millennium.

Imam Nawawi (631-671 H/1233-1272 ad) discussed fully the subject of bone and teeth transplantation in his voluminous reference textbook of jurisprudence Al Majmooh (14) and his concise textbook Minhaj Attalibin.(15) Al Imam Asshirbini commented on this subject in his book "Mughni Al Muhtaj".(16) Zakaria Al Qazwini (600-682 H/1203-1283 AD) advocated the use of the porcine bone graft as they take much better than Xenografts and function more efficiently,(17) despite the fact that Muslims consider the pig and its products untouchable. Jurists allowed the use of porcine material in medicine provided there was no other equal alternative.

Some Islamic Principles and Rules that are Related to Organ Transplantation

1. Islam considers disease as a natural phenomenon. It is not caused by demons, stars or evil spirits. Indeed, disease is not even caused by the wrath of God or any other celestial creature. Diseases and ailments are a type of tribulation which expiates sins. Those who forebear and endure in dignity are rewarded in this world and the Day of judgment.
2. Man should seek a remedy of his ailment. The Prophet (PBUH) told the Muslims to seek remedy and treatment.(18) He ordered his cousin Saad ibn Abi Wagas tó seek the advise of Al Harith ibn Kaledah, a renowned physician of his time.(19)
3. The Prophet (PBUH) declared there is a cure for every illness, though we may not know it at the time.(19)
4. We are encouraged to search for such a cure.(20) New modalities of treatment should be searched for and applied if proven successful.
5. The Prophet ordered Muslims to be compassionate to every human being. He also said "All mankind id the family ( ) of Allah; Those who best serve his family are best loved by God."
6. The human being should always keep his dignity even in disease and misfortune. The human body, living or dead, should be venerated likewise.
7. However, doing postmortem or donating organs from a cadaver does not mean mutilation of the corpse or an act of disrespect. The harm done, if any, by removing any organ from a deceased should be weighed against the benefit obtained, and the new life given to the recipient. The principle of saving human life takes precedence over whatever assumed harm that would befall the corpse.
8. In the case of a living donor, the principle of doing no harm "premium non nocere" is invoked. The donor cannot give one of his vital organs, which would end his life. It is an act of homicide or suicide, both of which are considered among abominable detestable crimes in Islam.

   Donation of an organ whose loss would usually cause no harm or a minimal increased risk to the health or life of the donor is acceptable. It invokes the principle of accepting the lesser harm when faced with two evils. The harm done by the disease, which can kill a human life, is not to be compared to the harm incurred by donation.
9. Organ transplantation is a new modality of treatment that can save human lives and improve the quality of life to many others. Islam encourages search for cure & invokes Muslims not to despair, for there is certainly a cure for every ailment, albeit we may not

know of at the present time.

10. Donation of organs in an act of charity, benevolence, altruism and love for mankind. God loves those who love fellow humans and try to mitigate the pains and sorrows of others and relieve their misfortunes.
11. Good intention: any action carried with good intention and which aims in helping others is respected and indeed encouraged, provided no harm is done.
12. The human body is the property of God, however man is entrusted with his body, as well as with other things. He should use it in the way prescribed by God as revealed by His messengers. Any misuses will be judged by God in the Day of Judgment, and transgressors will get their punishment. Suicide is equated in Islam with homicide. Even cremation of the corpse, which should be done as soon as possible.
    Donation of organs is not an act of transgression against the body. On the contrary, it is an act of charity and benevolence to other fellow humans, which God loves and encourages.
13. The human organs are not a commodity. They should be donated freely in response to altruistic of brotherhood and love for other fellow men.

Islam Jurists Fatwas (Decrees) Regarding Organ Transplantation

Muslim surgeons practised autograft transplantation which they learned from other nations, especially Indians. They also practised teeth and bone grafting from both animal and human sources (i.e Xenograft, and Homografts) since one millennium, after obtaining the consent of the jurists.

In the twentieth century, the Muslim jurists sanctioned blood transfusion, though blood is considered as "Najas" i.e. dirt. The Fatwa of the Grand Mufti of Egypt No.1065 dated June 9th, 1959, is an example of Islamic jurists attitude to new modalities of treatment.(21)

The majority of the Muslim scholars and jurists belonging to various schools of Islamic law invoked the principle of priority of saving human life and hence gave it precedence over any other argument. Sheikh Hassan Maamoon (The Grand Mufti of Egypt) also sanctioned corneal transplants from cadavers of unidentified persons and from those who agree to donate after their death (Fatwa No. 1087, dated April 14th, 1959).(22) His successor, Sheikh Hureidi extended the Fatwa to other organs in 1966 (Fatwa No. 993).

Sheikh Khater the Grand Mufti of Egypt issued a Fatwa (Decree) allowing harvesting of skin from unidentified corpses in 1973.(24) Grand Mufti GadAlHaq sanctioned donation of organs from the living provided no harm is done and provided it is given freely in good faith and for the sake of God loving and human fraternity. He also sanctioned cadaveric donors provided their is a will of testament or the consent of the relatives of the deceased. In case of unidentified corpses, an order from the Magistrate should be obtained prior to harvesting organs (Fatwa No. 1323, dated December 5, 1979).

The Saudi Grand Ulema Fatwa No 99, 1982 addressed the subject of auto grafts which was unanimously sanctioned. It also sanctioned with a majority of votes, donation of organs both by the living and the dead, who gave a will of testament or the consent of the relatives.(26)

The Kuwaiti law No. 7, 1983 reiterated the previous Fatwas and pointed out that the living donor should be over 18 in order to accept his informed consent.

The subject of "Brain Death" was not addressed in all these Fatwas. It was discussed for the first time in the Second International Conference of Islamic jurists held in Jeddah, in 1985. No decree was passed then, until further studies and consultations were obtained. In the Third International Conference of Islamic jurists held in Amman in 1986. the historical resolution (No.5) was passed with majority of votes, which equated brain death to cardiac and respiratory death.(27)

Death in the true Islamic teachings is the departure of the soul, but since this cannot be identified, the signs of death are accepted. This decree paved the way for extension of organ transplant projects which were previously limited to living donors. Campaigns for organs donations from brain dead persons were launched both in Saudi Arabia and Kuwait. The unfortunate high incidence of motor vehicle accidents in the Gulf area provides many cases of brain death.

This tragedy should be averted by using and pursuing stricter traffic laws and other means. Meanwhile it is a great pity to waste such candidate cadavers without trying to save the life of many others who need these organs. The Islamic League conference of jurists held in Mekkah Al Mukaramah (December 1987) decree No. 2 10th session, did not equate cardiac death with brain death. In fact, it did not recognize brain death, as death. However it sanctioned all the previous Fatwas on organ transplantation. This decree had little publication in the media, and the authorities in Saudi Arabia seem to ignore it. Cardiac and kidney transplants from brain dead individuals continued without any hindrance from the jurists.

The most detailed Fatwas on organ transplantation was that of the fourth conference of the International Jurists held in Jeddah, February 1988 (resolution No. 1). It endorsed all previous Fatwas on organ transplantation, clearly rejected any trading or trafficking of organs and stressed the principle of altruism.(28)

The jurists started to discuss new subjects related to organ transplantation viz; a) transplantation of the nervous tissues as a modality for treating Parkinsonism or other ailments. b) transplantation from anencephalics, c) transplantation of tissues from embryos aborted spontaneously, medically or electively, d) left over pre-embryos from IVF projects.(29)

It sanctioned transplantation of nervous tissue to treat ailments such as parkinsonism, if this modality of treatment proves superior to other well established methods of treatment. The source of the nervous tissue may be:

a) The suprarenal medulla of the patient himself (autograft).
b) The nervous tissue from an animal embryo himself (xenograft).
c) Cultured human nerve cells obtained from spontaneous abortion or medically indicated abortion.
d) Human nervous tissue obtained from spontaneously aborted fetuses or medically indicated abortions.

However it deplored doing abortions for the sake of procuring organs. it reiterated the Islamic views against elective abortion which is only allowed to save life or health of the expected mother.

If the fetus is viable then measures should be taken to resuscitate it and preserve its life. If, however, the aborted fetus is not viable, organs can be produced if the parents donate and only when the fetus is declared dead.

The aborted fetus is not a commodity and commercialism is not allowed.

The anencephalics can not be used as organ donors until declared brain dead. The full informed consent of the patient should be obtained in every case.

As regards left over Prembryoes from IVF projects, the jurists recommended that only the needed ova should be fertilized by the husband's sperms. However if excess fertilizer ova were found, they should be left to die spontaneously. Cryo preservation or donation of these fertilized ova is not allowed.

The jurists also discussed transplantation of genital organs. It did not allow the transplantation of gonads, as they carry all the genetic inheritance from the donor. However, they sanctioned the transplantation of the other internal sex organs.

New Frontiers are opened and the Islamic jurists are keeping pace with the tremendous advancements in medicine and technology.

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UK Shariah Council approves organ transplants

The Muslim Law (Shariah) Council in the UK has announced that it is acceptable for Muslims to donate organs and to recieve transplants. The statement, which has been agreed by Sunni as well as Shia scholars, was made after 2 years of deliberations. The discussions had been initiated by transplant coordinators in the Midlands concerned about how Muslims tended to perceive that organ donation was prohibited by their religion. The Council has resolved that:

The medical profession is the proper authority to define the sign of death; Brain-stem death is a proper definition of death and constitutes the end of life for the purpose of organ transplantation; The Council supports organ transplantation as a means of alleviating pain or saving life;

Muslims may carry donor cards; in the absence of a donor card or an expressed wish of the deceased to donate organs, the next of kin may give permission for organ donation;

organ donation must be given freely without reward. The statement has been welcomed by transplant co-ordinators. The high incidence of hypertension and diabetes amoung people from the Indian subcontinent (Asians) has meant a high prevalence of renal failure among those groups. In 1991 in the West Midlands Asians formed 9.7% of the population but 18.7% of those on the waiting list for kidney transplants. In addition, although 6% of organs donated are from group B donors. 34% of Asians belong to that group. An Asian needing a kidney has to wait an average of 2 years, compared with 4-6 months for white Britan

Source: Viven Choo Lancet, Vol. 346, July 29, 1995, Pg. 303

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Practitioner's Column

Allergy and Chronic Allergic Rhinitis

Mir Mohammad Maqbool*

JK Practitioner 1996; 3:2 109-110

The word allergy means hypersensitivy to a specific substance. Allergen means a substance supposed to produce symptoms of allergy.

The earliest written mention of an allergic disease was an account of how king Meves of Egypt was fatally stricken by the sting of a wasp in 2641 B.C. In Medical Science allergy as a speciality in only sixty years old, but in this short span of time great progress has been made in the application of new scientific approaches to the mechanism involved in the allergic response:-

- The role of antigen
- Antibody reaction
- The diverse forms of clinical allergy
- The many aetiologic factors associated with the allergic state and
- The development of symptom relieving antihistamincs and antiserotenines.

Allergens can trigger an allergic reaction by:

1- **Ingestion,** i.e. by eating or drinking something one is sensitive to.

2- **Inhalation,** i.e. through the air we breath. Such allergies are increasing because of increasing airpollution.

3- **Contact,** e.g. clothes, cosmetics.

Allergic Symptoms:

In the gastrointestinal tract, diarrhoea, vomiting and colic.

In the respiratory tract : sneezing common cold and asthma.

One the skin: rashes, swellings, itching and exudation. The first step in controlling allergy is to identify the allergen.

ANYTHING UNDER THE SUN, INCLUDING THE SUN ITSELF,CAN CAUSE ALLERGY.

The following checklist of allergens is designed to help the allergic patients to identify the factor/s that he is sensitive to.

CHECKLIST OF ALLERGENS:

Acetone, Alcohol, Antibiotics, Antibites, Asbestos, Aspirin, Aroma, Bacteria, Bee stings, Beverages, Blood transfusions, Boric acid, Brinjals, Chemical, Champhor, Curds, Cosmetics, Cocoa, Crabs, Creams, Cereals, DDT, Detergent, Drugs, Dust, Dyes, Eau-de-cologne, Eggs, Elaichi, Ether, Fibres, Feathers, Fish, Flowers, Foods, Fumes, Furs, Foams, Garlic, Gasoline, Gelatine, Grapes, Grass, Guavas, Hair, Haldi, Hay, Honey, Household pets, Husk, Horse hair, Ice, Ice-cream, Inks, Insecticide, Insects, Iodine, Jackfruit, Jaggery, Jasmine, Jeera, Jute, Kajal, Kerosene, Kesar, Kumkum, Lady finger, Lily, Latex, leather, Lemon, Lipstick, Lobster, Mascara, Meat, Metals, Milk, Mosombi, Mustard, Mud, Nailpolish, Naphthalene, Nuts, Nylon, Oats, Oils, Onions, Oranges, Oysters, Perfumes,Peppermint, Paint, Pencillin, Plants, Plastics, Pollen, POmades, Prawns, Quill, Quilt, Quinine, Radiation, Rayon, Resins, Roses, Rubber, Silk, Spices, Sulpha, Sun, Shellfish, Synthetics, Shrimps, Seera, Soap, Talc, Tobacco, Tomato, Toothpaste, Turpentine, Terylene, Ultra-violet rays, Urea, Urethane,Vinyl, Vaccination, Vanila, Varnish, Vaseline, Wasp, Wax, Weather changes, Weeds, Wheat, Wool, X-rays, Xylene, Xylocaine, Yam, Yeast, Yew, Yolk, Zinc oxide, Zygospores,

Repeated attacks leave allergy victims prone to respiratory infections and sometimes scar the respiratory tract. Allergies can even be fatal. Each year thousands die of asthma, a respiratory disorder that is often caused by allergy, and at least 50 from insect stings that bring on anaphylactic shock.

Doctors are still puzzled by certain aspects of allergy-Injections of allergy victims, build some tolerance to those substances, but just how, is not known. The shots are rarely given for allergies to foods or animals. Although antihistamines often help relieve symptoms, their effectiveness varies from one person to the next. Within the decade, research may yield drugs that will prevent the reaction entirely or at least relieve the symptoms better than anything now available.

Out of all allergic conditions, chronic allergic rhinitis is most common in Kashmir.

The nose acts as a filter for suspended particles in inhaled air and great majority of all particles are either deposited or breathed in and out. Repeated exposures of the nasal mucosa to allergens results in a condition of hypersensitivity where inhalation of particles triggers an allergic response with symp-

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*Ex-Professor & Head
ENT Deptt. Medical College Srinagar
Kashmir, India.

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toms of -nasal congestion, -itching, -hypersecretion. Presenting as intense irritation in nose and eyes associated with sneezing, nasal obstruction, profuse watery discharge and excessive watering of eyes.

Chronic allergic rhinitis may be:-

(i) Seasonal
(ii) Perennial

Seasonal:- allergy depends upon climate and environment and its peak months vary from place to place. Besides nose, conjunctive may appeared and bronchial mucosa may also be involved showing attacks of bronchospasm. The nasal mucosa appears swollen and pale or may have a bluish tinge and airway is diminished.

Perennial type:- allergy can occur anytime during the year and symptomatology is similar but not so marked as in the seasonal type.
It may be due to:-(i) Inhalant substances like house dust, smoke, spores, etc. or(ii) Ingestants like milk, egg, fish and cheese of drugs, or bacteria or (iii) Contactants like clothes, perfumes and cosmetics.

Pathology:

When allergen comes in contact with the sensitised mucosa, there occurs Antigen-Antibody interaction on the surface of mast cells and basophil leucocytes, present in the nasal secretion, resulting in release of mediators such as histamine and other Kinins which are responsible for the symptoms of allergic rhinitis due to both the i) local effects on glands and blood vessels and ii) Reflex actions by stimulating sensory nerve endings.
Because of oedema and blockage secondary bacterial infection may occur. Polyp formation in the nose and sinuses and frank sinusitis may occur as the disease progresses.

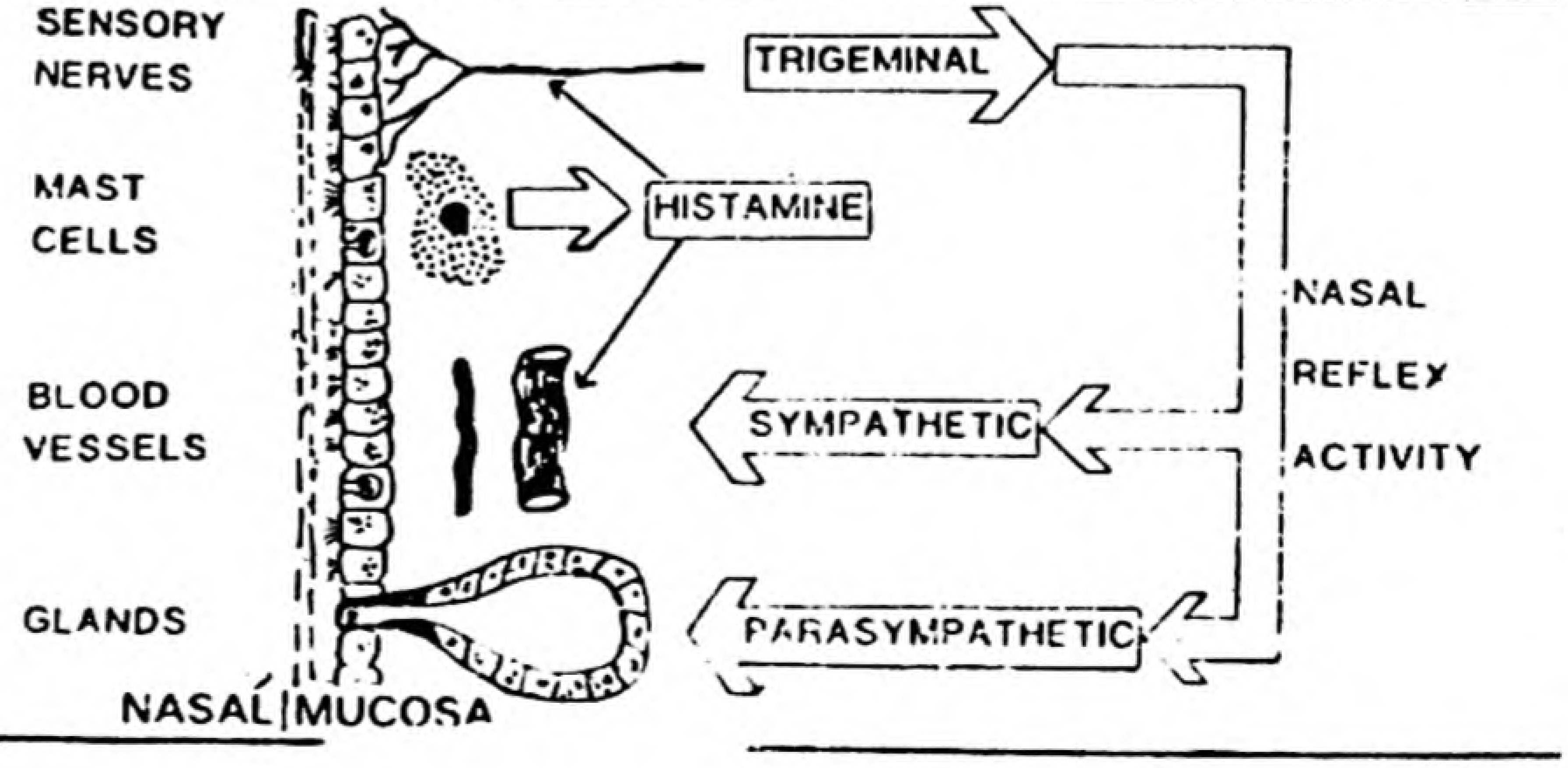
There are certain diagnostic clues to allergy found in the children or young adults suffering from chronic allergic rhinitis which can often be recognised as much by keen clinicians.

i. The allergic shiners found as dark areas under the eyes as a result of discoloration in the lower oribitopalperbral grooves caused by venous stasis.
An additional factor is spasm of Muller's muscle, a smooth muscle that is the only involuntary eyelid muscle, this muscle impedes venous return in the skin and subcutaneous aveolar tissue of the lower eyelids so that in addition to discoloration, oedema occurs, resulting in "bags" under the eyes.

ii. The transverse nasal crease is another visual sign. This appears in children as a horizontal hypopigmented or hyperpigmented groove across the lower third of the nose where the bulbous, soft portion meets the more rigid nasal bridge. It results from constant rubbing of the itching obstructed nose and takes at least two years to develop. The allergic crease disappears when the tip of the nose is pulled downward. This fact differentiates it from the familial transverse nasal groove.

iii. The allergic salute The often dripping nose is being wiped off by the children with hand. The thenar eminence is rubbed against the tip of the nose with rest of the hand stretched out as in salute.

iv. Long, thin, silly eye lashes are usually seen in young girls suffering from chronic allergic rhinitis.



Local and reflex actions of histamine in the nasal mucosa.

HIV Infection In Children

G.H. Hajini*

JK Practitioner 1996; 3:2 111-112

WHO case definition of AIDS in children:
Paediatric AIDS is suspected in an infant or child who presents at least 2 major and 2 minor signs in absence of any known cause of immuno suppression.

Major signs: 1. Weight loss/ Failure to thrive, 2. Chronic diarrhoea of more than 1 month, 3. Prolonged fever of more than 1 month.

Minor signs: 1. Generalized lymphadenopathy, 2. Orophrengeal candidiasis, 3. Repeated common infections, 4. Persistent cough for more than 1 month, 5. Generalized dermatitis, 6. Confirmed maternal HIV infection.

Transmission:
Most of the children infected with HIV acquire it from their mothers. Perintal transmission (mother to child) can occur in one of the three ways.
a. Tranplacental b. Intrapartum. c. Breast feeding. Relative frequency of each of the three routes is unknown. HIV has been isolated from cord-blood, amniotic fluid and foetal material obtained from abortion. HIV can be present in the secretion of female genital tract of infected women and thus contact of an infant with maternal blood during delivery is a potential risk of infection. HIV has also been isolated from breast milk and this can also be a source of infection. But woman whose infectious status is unknown is advised to breast feed in areas where infant mortality from other cause is more.

Parental transmission:
In places where screening of donors for HIV infection is not possible, blood transfusion continues to be an important route of HIV transmission to children. The role of contaminated injections is unclear but their contribution is probably small.

Other modes of transmission:
Unlike hepatitis B virus infection has never been attributed to be transmitted through routine close contact, even after biting. Children with HIV infection should not therefore be excluded from communities (family, day-care schools) unless their behaviour is agressive.

Frequency of Perinatal transmission:
Most of the studies estimate it to be around 25% (7-60% range). The survival of children born of HIV seropositive mothers varies. The mortality of such infants was about 5% at 15 months of age in Europe and America whereas in Africa it was 12-39% at the age of one year. The main causes of death in such children included prematurity, diarrhoea, pnenmonia, AIDS and meningitis. Higher morbidity and mortality rates in Africa are perhaps due to:
a. Higher rate ofperinatal transmission.
b. Increased risk of infection from environment.
c. Poor nutrition.
d. Less access to standard medical care.
e. Non availability of specific therapy including immunoglobulins, anti-viral and anti-retroviral drugs.

Clinical features:
Incubation period between the birth and the appearance of first clinical manifestation, varies from few months to several years. Many of the clinical features are not specific of HIV infection and are seen in other diseases like tuberculosis, Malnutrition, Malaria and Measles. Important characteristic in HIV infections however is that these are often persistent, recurrent and show no response to existing therapy, Children who fail to thrive or have diarrhoea, fever and respiratory distress have a poorer prognosis compared to those with generalized lymphadenopathy, hepatosplenomegaly or parotitis.
Common clinical features are summarized as:
Generalized lymphadenopathy, Hepatomegaly, Splenomegaly, Chronic parotitis, Thrush, Chronic cough, Chronic recurrent diarrhoea, Chronic fever, Chronic dermatitis, Weight loss, Protein energy malnutrition, Pneumonia.
Severe and recurrent bacterial infections frequently observed in such children are usually due to Salmonella, Mycobacterium tuberculosis, Streptococcal pneumonia.
AIDS encephalopathy is increasingly been recognized in children and is charasterized clinically by progressive loss of developmental milestones, acquired microcephally, ataxia, pyramidal signs, seizures and coma. Such children have poor prognosis.
Opportunistic infections are usually fatal.

***Prof. & Head Deptt. of
Dermatology & Venerology
Medical College Srinagar Kashmir, India.**

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Difference between AIDS in Children and Adults:

	Children	Adults
1. Interstitial pneumonitis and chronic parotitis	More common	Less common
2. Severe, recurrent bacterial infectious	Major problem	Comparatively less
3. Tuberculosis	Less prevalent	More Prevalent
4. CNS opportunistic infection and toxoplasma	Uncommon	More Common
5. Kaposis sarcoma and CNS Lymphoma	Rare	More Common
6. HIV encephalopathy	More frequent	Less frequent
7. Hypergammaglobulinemia	More pronounced	Less pronounced

HIV-2 infection in Children:
In west Africa, HIV-2 is emerging as a new causative agent of AIDS. The perinatal transmission rate is however lower than that of HIV-I, clinical spectrum however does not differ significantly from that of HIV-I infection.

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Students Page

Cosmetics In Practice

Sheikh Manzoor*, Shugufta Parveen*

JK Practitioner 1996; 3:2 113-115

Introduction

Body image is largely cutaneous and may be modified by many factors, such as clothing, hair style, tattooing, scarification, and body painting, The 20th century equivalent of body painting in what is generally assumed to be the civilized western world is cosmetic usage.

The cosmetic industry employees the advertising industry to help sell its products. The advertising industry which is considered to be largely male dominated, projects certain fairly limited images of women, targeted mainly at the female magazine reading public but also at film and television audiences.

The most desirable values projected in this market place situation include a youthful, wrinkle-free, odorfree, greesefree, spotfreex and preferably hairfree skin with the exception of the scalp which should have a luxuriant growth of hair. Ideally there should be no hair on the face, arms, axillae, legs, chest, abdomen, or even the bikini area. Further more, the ideal today is portrayed as willowy thin inconspicuous breasts. In short, the image the advertising industry is projecting is indicating to women that they should be almost entirely prepubertal in their cutaneous desires, and so most secondary sex characteristics are seen as frankly undesirable.

Not only should be cutaneous image of a mature women be baby like, but the advertising industry also projects infantile and often medieval ideas about skin and about its anatomy, physiology and skin care in particular. Thus the idea that skin breaths, or need to breath, is common place in cosmetic advertisements, and the dire consequences that are supposed to follow if a pore is "clogged" are also perpetually advanced.

The idea that the skin needs regular cleansing and feeding is also interminably stressed. The view that the skin needs regular cleansing goes back to atleast biblical times, but even William Shakespare had his doubts about the therapeutic efficacy of cleansing in acne when he wrote:

> "THAT'S A FAULT THAT WATER WILL MEND
> NO SIR, IT IS IN THE GRAIN: NOAHS FLOOD
> COULD NOT DO IT"

Finally, is it possible that the desirable prepubertal image that is dangled before women could be playing a part in the pathogenesis of body image disturbances, such as those seen in patients with dermatologic non disease and women with the anorexia nervosa.

Definition

"Cosmetics are articles intended to be applied to the human body for cleansing, beautifying, promoting attractiveness or altering the appearance without affecting the body's structure or functions. Included in this definition are products such as skin creams, lotions, perfumes, lipsticks, finger mail polishes, eye and facial make-up preparations, shampoos, permanent waves, hair colour, tooth-pastes, deodorants and any ingredients intended for use as a component of a cosmetic products. Soap products consisting primarily of alkalis act as fatty acid and making no label claim other than cleansing of the human body are not considered as cosmetic".

Products that are cosmetics but are also intended to treat of prevent disease or affect the structure or functions of the human body are considered drugs under cosmetic provisions. Examples include "fluoride" toothpaste, hormone creams, sun....... preparations intended to protect against sunburn, antiperspirants that are also deodorants and antic and ruff shampoos. Most cosmetics that are also drugs are over the counter drugs. The requirements for drugs are more extensive than those for cosmetics. Drugs have to be proven safe and effective before being marketed.

COSMETIC TERMINOLOGY:

1. **Cleansers:** available in liquid, solid, bar, granular, and other formulations. Many have detergent - type ingredients and can be drying or irritating, oils

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*Deptt. of Dermatology
GMC Srinagar Kashmir 190 010

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are added to cleansers promoted for dry skin.

2. Astringents: Many contain alcohol, with hazel and fragrance. These are promoted to close the pores after washing. After shave lotion is usually an astringent.

3. Moisturizing and creams: Emollients applied to dry skin, aging skin or ashy skin the fine whitish scale of exfoliating epidermis complained of by many black patients. Many of these products are comedogenic.

4. Foundation or base make-ups: Any pigmented product intended for application to the face and throat areas to colour the skin evenly and hide facial imperfections such as scars, depressions and hyperpigmentation or hypopigmentation areas. They come in various forms: lotion creams, pressed powders etc. Foundations are loosely divided into oil in water emulsions (oil based) water in oil emulsions (water based) and solvent based (oil free) categories. The total oil phase of a liquid make up can range from 10-40% of the total formulations.

5. Powders: Applied over foundation to give a natural texture to the skin. Available in "loose" and "pressed" forms. If pressed, the powder may be pressed with mineral oil or isopropyl mysistate both known comedo promoters.

6. Blushers: This product is used to impart colour to the cheek area, it is the old-time "rouge" and is available in various formulations from dry powder to cream. The D&C rid pigments used within have been found to be comedogenic, whereas carmine and ultracarmine blues are not. This may explain why cosmetic acne is frequently most severe on the upper cheek bones where these blushers are applied.

7. Hypoallergenic: This simply means that the manufacturer thinks that the product has fewer allergens than most cosmetics.

Colour used in Cosmetics

- Aluminum powder, - Annatto
- Bismuth citrate, - Bronze powder
- Caramel, - Caramine
- Carotene, beta
- Chrominum hydroxide green
- Chromium oxide greens
- Chopper powder, - Dihydroxyaccetone
- Disodium EDTA - copper
- Ferric ammonium ferrocyamide
- Ferric ferrocyamide, - Guanine
- Guaiazulene, - Henna
- Iron oxide, - Lead accetate
- Magnesium violet, - Mica
- Potassium Na copper chlorophyllin
- Sliver, - Titanium dioxide
- Ultra marines (Blue, green, pink, red, violet), - Zinc oxide.

Preservations

They are used to control microbial contents, to prevent deterioration of the product, and to extend shelf life. The various preservatives used are:

- Benzonic acids (or salts)
- Benzyle paraben
- Boric acid
- Bulyl paraben
- Chlorhexidine digluconate
- Dehydroacetic acid
- DMDM hydantoin
- Ethyl paraben
- Formal dehyde
- Glutaral
- Imidazolidimyle urea
- Methyle isothisazolinone
- Methyle paraben
- O-Cyummene 5-cl
- Phenyle mercuric accetate
- Propyl paraben
- Quaternium-15
- Sodium pyrithone
- Scorbic acid.

Non prescription skin products

Following is a list of products often times advertised as offering protective capacillities for the skin:

- Allantoin
- Calamine
- Cocca butter
- Dimethacone
- Glycerin
- Kaolin
- Petrolatum preparation
- Shark liver oil
- Sodium Bicarbonate
- Zinc oxide.

Products for dry and mature skin

The following products are offered to the public as especially helpful for dry or aging skin:

- Collagen
- Urea,

- Hyaluronic acid
- Mineral oil
- Petrolatum
- Lanolin
- Lanolin alchol
- Lanolin oil
- Acetylated lanolin alcohol
- Dimethacone
- Stearic acid
- Isostaric acid
- Myristic acid
- Linoleic acid
- Vegetable oil
    - Sesarne oil
    - Almound oil
    - Avacodo oil
    - Olive oil
- Hydrolyzed animal protein
- Milk protein
- Amino acids.

The range of cosmetics and their usage:
Cosmetics are used to enhance the appearance of the body, to clean it, to impart a pleasing smell or mask an unpleasant one, or as a fashion accessory now-a-day some cosmetics are not being marketed with the claim that they have an active ingredient for example one which can reverse aging.

Site	Products
Skin	Moisturiser, cleanser, soap, make up remover, powder, rouge foundation, tonic, perfume, after shave, bath additive, sun screen.
Hair	Shampoo, conditioner, bleach, colorant,permanent waving, straightening,lacquer, gel hair, removing agents.
Eye lids	Mascara, eyeshadow, eyeliner, pencil.
Nails	Nail varnish, varnish remover.
Lips	Lipstick, lip gloss, sunscreen.

Reactions to cosmetics
Side effects are comparatively rare when the vast usage of cosmetics is considered but, none the less, 12% or more of adults have had a reaction to a cosmetic. Some responses e.g. stinging with after shave due to the alcohol base, are expected and do not constitute a reaction. The preparations most likely to cause a problem are eye and facial cosmetics, anti perspirants and deodorants, hair colorants and soaps. Reactions can be categorized as follows:

Cosmetics	Reaction
Soap, detergent	Mostly irritant
Deodorant, antiperspirant	Irritant somedays allergic
Moisturer	Irritant and allergic
Eye shadow	Mostly irritant
Mascara	Mostly irritant
Permanent wave	Irritant and allergic
Hair dye	Allergic
Shampoo	Mostly irritant

Irritant contact dermatitis
This is particularly seen in atopics and those with a sensitive, skin, soaps which are drying and alkaline (the normal Ph of facial skin is about 5.5) and deodorants cause mostly irritant dermatitis. Lanolins, detergents and preservatives may also be irritant.

Allergic contact sensitivity
This mostly commonly, develops to fragrances preservalives, dyes, (e.g. parphenylene diamine-PPD) lanolins & glyceryl monothioglycolate. The eruption usually develops at the place of application of product (usually the face), but this is not always so, as substances can be transferred to another site where they cause symptoms. For example contact allergy to sulphonaimide. Formaldehyde resin in nail varnish most often manifests as an eruption around the eyelids or on the neck.

Contract urticaria and other adverse reactions
Contact urticaria presents as a wheal and flare response within a few minutes of the applications of a substance. It may occur with compounds in perfumes, shampoos and hair dyes. Other adverse reactions include nail dystropties caused by nail cosmetic use, hair breakage and weathering due to improper use of permanent waving, hair straitness or dyes, pigmentatives and acne.

Management of the cosmetic reactions
A patient intolerant of a cosmetic should stop, the use of all cosmetics and if necessary be treated with a topical steroid until the reaction subsides. All the cosmetics and preparations that have been used must be examined, and patch testing performed if appropriate. Alternative cosmetics can be used, but kept to a minimum.

Clinical Images

JK Practitioner 1996; 3:2 116

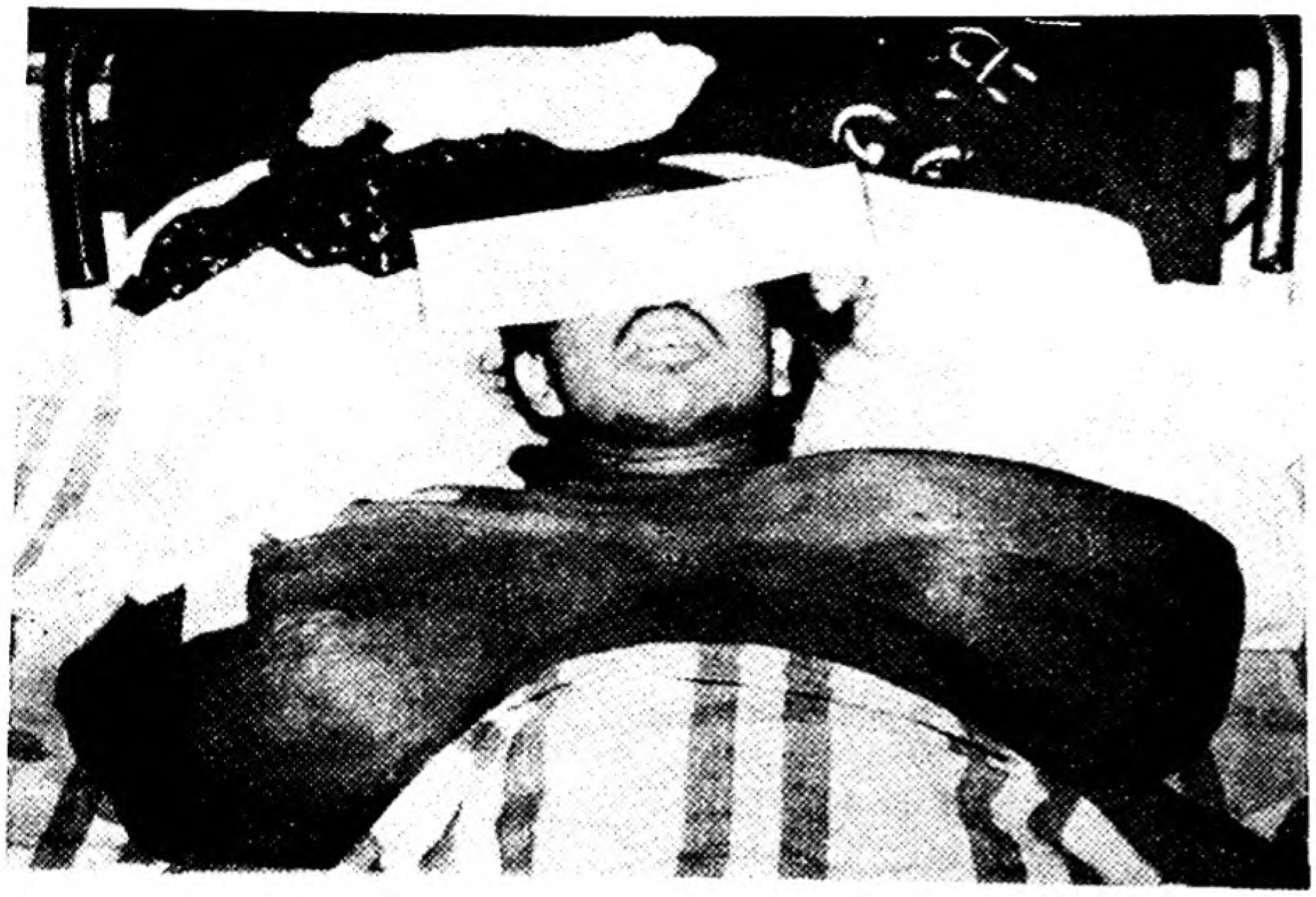

Fig 1:- Breasts (Both)

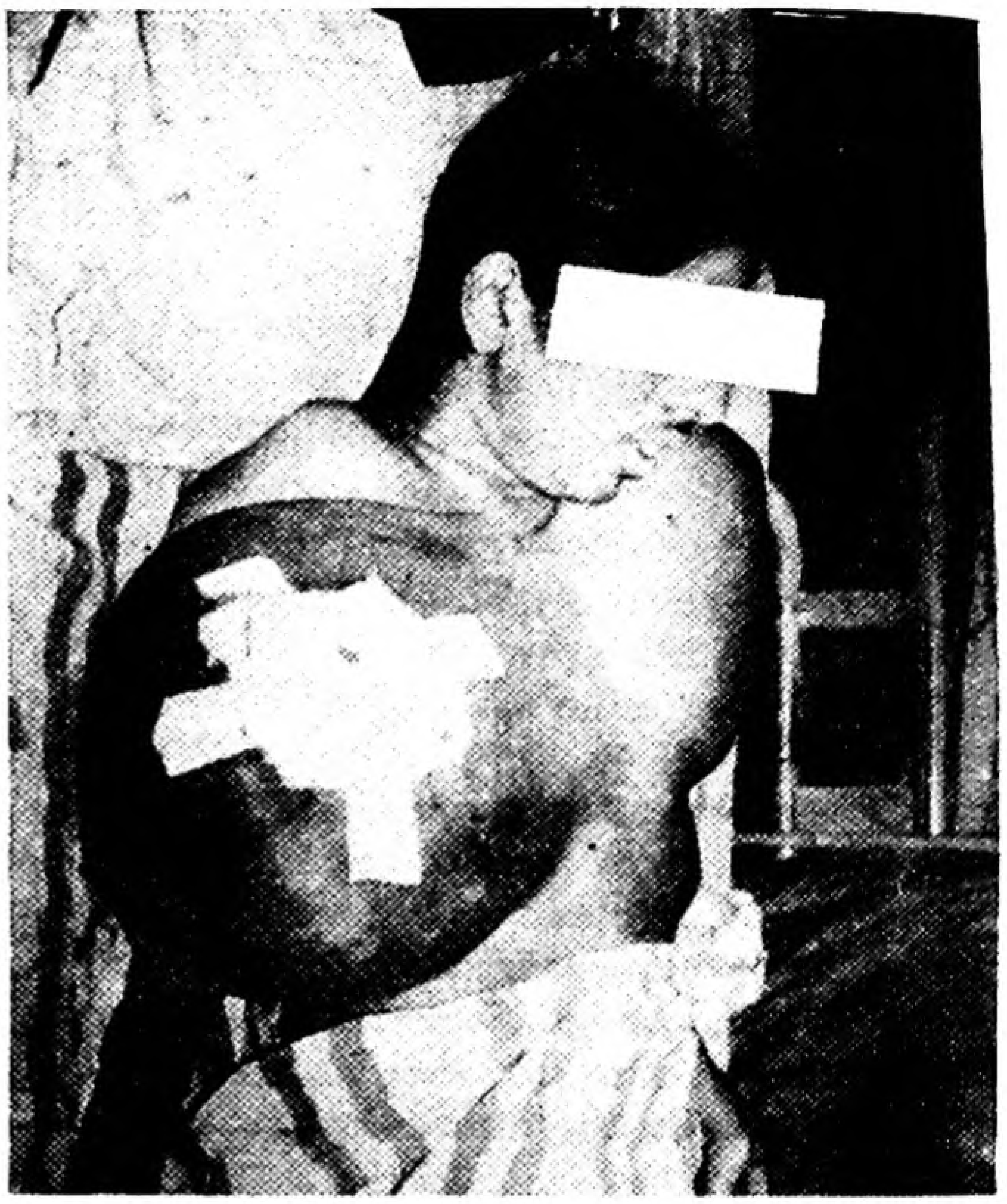

Fig 2:- Right Breast (Laretal view)

Summary of Case

A 35 year old female noticed progressive enlargment of breast starting around 10th week, they assumed massive size and caused discomfort to the patient because of weight. Her weight was 43 Kgs, ultrasonography at 16 weeks confirmed pregnancy. She had nutritioal anemia, hormone assay in the form of follicule stimulating hormone, lutinizing harmone, growth hormone and prolactin were normal. She was given Danazol 400 mg orally per day for 20 days. There was no decrease in the size. Total mastectormy was done. Both breasts weighed 11.5 Kgs and 2 litres of pus drained from Rt breast. There was no recurrence for 2 years of followup.

Diagnosis:- Gestational Macromastia

Authors :
* A. H. Zargar, * B. A. Laway, * S. R. Masoodi, * N. A. Shah, * M. A. Darzi, A. H. Wani, F. A. Bhat.

* Deptt of Endocrinology and Plastic surgery, SKIMS, Srinagar, Kashmir, India.

MODERN ENDOSCOPY CENTER, JEHLUM MARKET, BATAMALOO, SRINAGAR.

Performance Appraisal

- A Subjective Phenomenon in Hospitals

*M.A. Wani

JK Practitioner 1996; 3:2 117-118

Hospitals are highly developed and socially valued institutions of our health care system. They have a long and rich history of providing primary, secondary, tertiary and quarternary Medicare to thousands of needy patients. While delivering various patient care services hospitals need a huge work force in terms of medical para-medical, nursing and non-medical personal. This work force commonly termed as human resources utilize almost two thirds of hospital budgets on an average basis.

We know that hospital resources are limited whereas costs and demands in terms of patient care services are rising day by day, as such the containment of patient care service costs become need of the hour. To achieve this quality, efficiency and optimal utilization of the available work force become of vital importance. The same can be achieved by various managerial techniques and activities. One such activity which promises to increase motivation and performance of human resource component, is by advocating meaningful I.P.R. system.

Performance appraisal is probably one of the oldest yet still most imperfect of managerial activities. People have been making informal appraisals of each others performance for about as long as the human race has indulged in group activities. However, interest in informal appraisals dates back to world war Ist when Americans started evaluation of managerial jobs with the assumptions that high morale of managers can lead to high productivity of organizations. With the passage of time the evaluation involved all categories of jobs but was limited to the practice of appraising personality only. Between 1950 and 1960's a switch away from appraising personality towards job performance was seen where accent was on participative goal setting and problem solving. In 1970's these views were elaborated and stress was given on openness and subordinate participation in the appraisal process with the rise of trade union interests in the appraisal practices. Today individual performance reviews (IPRF's) commonly known as APR's are more result-oriented and based stressing on current rather than future performance.

Performance appraisal is the assessment of how a staff member is doing the job. Assessing is what a supervisor or team leader does to determine how close his/her staff comes to achieve work objectives as specified in their job descriptions.

IPR can define as any procedure which helps in collecting, checking, storing, giving and using of information collected from and about people at work for the purposes of adding to their performance at work. It enables employees to discover their strength and weakness so that they can minimize or correct their weaknesses. It also focusses on carrier development and training needs of individual workers leading to improved work performance. With reference to any ideal hospital service organization, it serves as a basis for making comparisons between staff in deciding on promotions, grade revisions and also at times acts as a motivating mechanism for improving performance and productivity by boosting morale and esteem of employees. It can also serve as a tool for collecting information for personal planning and management development. There is of course no one "right" way of operating appraisal schemes because there is no single perfect technique as greatest problem in writing APR's especially in hospital set up is the problem of subjectivity.

The problem of subjectivity:-

All appraisals have one or other form of subjective element due to following characteristics of managers/administrators.

Managers vary considerably in their ability and motivation to evaluate accurately the behavior of others.

The experience of appraise may affect the quality of appraisals.

Lack of information may result in subjective assessment. Here managers may be unfamiliar with the job requirements or duties of their subordinates or their subordinates due to physical distance.

Assessments may be based on scales that are too impressionistic and insufficiently tied to evidence. Mostly these impressions may be related to latest work performance of individual subordinates.

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*Medical Supdt. SMHS Hospital,
Medical College, Srinagar,
Kashmir - 190 010 (India)

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Halo Effect :-

Here qualities of individuals are judged very favorably (while writing APR's of friends, relatives). Conversally good qualities of subordinates may be masked resulting in back halo effect. (while writing for enemies, races etc.) In practice I have seen that assessment mostly depends on existing relation between managers and subordinates good relation for e.g. between friends, opposite sexes or otherwise assess subordinates favorably resulting in good remarks, assess subordinates favorably in good remark whereas poor relations assess subordinates critically resulting in adverse remarks.

Supervisors may feel that if they rate their subordinates unfavorably, this will reflect poorly on them as supervisor. the corollary of this is that good assessment will reflect favorably.

It has been seen in my experience that managers are compelled at times to rate their subordinates favorably. (by taking a lenient view of scales) in the belief that this will please their subordinates and improve their chances for promotions.

We know that subjectivity is a problem especially in such hospital Organizations where IPR system are confined and closed type. So it can be minimized by advocating a self appraisal system which involves subordinate participation. It can be done by giving the appraise a blank copy of the normal appraisal form so as to know his brief views about his job performance. But it is worth to mention here that appraises observations should be based on general rating of performance qualities rather than behavioral observation so as to avoid danger of objectivity. It would be followed by an interview between a manager and the subordinate so as to get a clear cut idea of the views of the appraises about his performance. The appraise era opinion should be given against this back-ground which would minimize subjectivity. there is clearly one type of self appraisal which holds great promise. This is when individual is asked to assess different, aspects of his or her job performance relative to one another rather than against the performance of his colleagues working on the similar post. The evidence here shows that subordinate judgments of this kind are more discriminating and accurate.

It has been seen in hospital organizations that relations between superordinates and subordinates are close as both are usually housed in one complex or at the most in adjusting complexes. As such while advocating a self appraisal system for hospitals care is to be taken about over ratings the performance of appraises which might force appraisers to take a lenient (favorable) view of the appraise, such over ratings if any, need to be discouraged at the time of interview or neglected at the time of final opinion with a view to implement a meaningful I.P.R. system.

Abbreviations :

IPR = Individual Performance Review.
ACR = Annual Confidential Report.

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Letters To Editor

Heriditary Orotic-Aciduria Can Be Caught

JK Practitioner 1996; 3:2 119-120

Introduction:
Books describing inborn errors of metabolism often make two remarks about these diseases (a) No treatment is of any avail or (b) No treatment is required. This often makes a study of these disorders unattractive for the general practitioner. To make matters worse often sophisticated laboratory tests are needed to diagnose these diseases. Heriditary tests are needed to diagnose these diseases. Hariditary orotic aciduria is an exception to this general rule. An alert House surgeon or even an Intern in a pediatric hospital can disgnose the disease from the clinical history and routine laboratory tests of blood and urine that are performed in every side room Lab. Indeed a simple urine glass (or any container for collecting urine) will prove to be the most important apparatus needed for the diagnosis of this disorder. Further this disorder can be easily treated and the patient made to live a normal life by giving uridine (150 mg/kg/day). Admittedly, it is a rare autosomal recessive disorder but the prospect of making an otherwise morbidly ill child live a normal life by simple treatment makes its diagnosis worth the effort. The purpose of this article is to draw the attention of practitioners and especially Interns, House surgeons and Consultants working in pediatric hospitals towards the clinical features of the disease and the simple tests that can be used to diagnose this interesting inborn error of metabolism. Aetiology ; Heriditary orotic aciduria occurs because of a deficiency of single protein associated with the enzyme activities orotate phosphoribosyl transferase and orotate decarboxylase. These enzymes catalyse an essential step in the biosynthesis of pyrimidines and the patient becomes dependent on dietary pyrimidines for normal life. This is exactly analogous to the single enzyme defect in the pathway of ascorbic-acid biosynthesis which makes man dependent on ascorbic acid in the diet. Thus like scurvy, Heriditary orotic-aciduria is a nutritional disease, which can be treated by providing an adequate amount of uridine in the diet. For patients with this disease pyrimidines become an essential dietary nutrient.

Clinical Aspects:
The clinical symptoms of Heriditary orotic aciduria are essentially those of pyrimidine deficiency. They are best appreciated by studying two case reports which have been taken from literature[1,2,3]

Case 1:
A normal child, was born to cansanguineous parents, in whom pallor was the first sign noticed at 3 months of age. At 6 months he was admitted to hospital for diahorrea. On making routine laboratory tests of blood he was found to have severe hypochromic megaloblastic anemia. Hemoglobin concentration was 6 gm%. R.B.C count 2.5 million/cumm. Anemia failed to respond to B_{12}, folic acid, pyridoxine, iron and ascorbic acid. Corticosteroids produced transient improvement. He had several episodes of uretheral and on one occasion ureteral obstruction due to crystalluria. Freshly voided urine on standing formed crystals (needle shaped on microscopic examination) which coated the walls of the container. They were identified as orotic acid. Patient showed dramatic reponse to concentrated yeast extract (containing 1.73g CMPO and 4.03g UMP) given dialy. The megaloblastic marrow reverted to normal after 22 days.

Case 2:
A normal child born to unrelated parents. Pallor was noted at 3 months of age. At 1 year he was so weak that he was unable to lift his head or sit without support. At 13 months he was found to be severely anemic with hemoglobin concentration 4.6 gm%. He failed to respond to iron, B_{12}, Pyridoxine and thyroxine. At 17 months he was underweight and failed to maintain a sitting position. He had Hb 8gm% HCt 25% and MCHC 32%. Peripheral red cells showed a reciculocyte count of 2%, peripheral smear showed marked anisocytosis and poikilocytosis. There were numerous macrocytes but other cells were microcytic. Smears also showed polychromatic cells, Howell Jolly bodies, cabots rings and nucleated red cells resembelling megaloblasts. There was Achlorhydria but normal serum B_{12} levels. Urine was perfectly clear when fresh, but when allowed to stand for several hours formed a large amount of crystals which adhered firmly to the wall of the container. The colourless fine needle shaped crystals were identified as Orotic acid. There was a dramatic reponse to uridine 1500 mg (150 mg/kg) given daily. The reticulocyte count rose to 5.4% and Hb to 11 gm%. Bone marrow morphology reverted to normal on 17th day of treatment. There was great improvement in general condition with

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weight gain of 3 pounds over a three week period and a spurt in the growth of hair and finger nails. At seven years of age height and weight were in 75th percentile. A single alternate convergent strabismus was still present with a small pigmented area in each sclera. He was somewhat mentally retarded (I.Q Wechslar Scale 73-81) with verbal skills superior to motor ones. He made slow progress at shcool.

Conclusion:

Although Heriditary orotic aciduria is a rare disease with only a handful of cases reported in literature yet because the children Hospital at Srinagar is the only pediatric hospital in the entire valley and caters to the entire pediatric population of the valley there are appreciable chances of a child with this error being referred to this hospital. The presence of a megaloblastic anemia in a child not responding to usual treatment like B_{12}, folic acid, pyridoxine and iron should alert the physician to the possibility of this disorder. The fact that urine when allowed to stand for several hours in the cold deposits needle like crystals of orotic acid which often adhere to the sides of the container and the dramatic response to uridine (150 mg/kg/day) should click the diagnosis although for confirmation the crystals must be sent to a specialised laboratory for identification. Although the disease often manifests itself in the first year of life a case has been reported where the symptoms first appeared at the age of 6 1/2 years, with proper treatment (uridine 150 mg/kg/ body wt/day) the person can live a fairly normal life although unfortunately some degree of mental retardation remains.

Syed Tafazul Hussain, M.Sc.
Asstt. Professor Biochemistry Deptt,
Medical College Srinagar, Kashmir, India

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Pulse Polio - Campaign in Kashmir Valley

Below are the targets achieved on account of Pulse Polio campaign in valley (1st dose & 2nd dose) from Dec. 95 to Jan 1996 for publication as given below:

S.No.	Name of District	Estimated Eligible Children 0-3 Years	O.P.V. Doses Given	
			0-3 Years	Above 3 Years
01.	Anantnag	75817	92273 (89571+2702)	12589 (12273+360)
02.	Budgam	47000	54636	4433
03.	Baramulla	81456	100850	117116
04.	Kupwara	36600	53920	4339
05.	Pulwama	45440	55176	4633
06.	Srinagar (Urban)	58660	48672	5293
07.	Srinagar (Rural)	23402	24027	2608
08.	Leh	7400	6921	1080
09.	Kargil	10057	9980	639
		385742	442914	44030

Dr. Manzoor A. Shawl
Director Health Services
Kashmir, India.

Meet The Professor

Dr. Mir Mohammad Maqbool
M.S; FICS

JK Practitioner 1996; 3:2 121

Permanent Address:
140 Karan Nagar, Srinagar Kashmir - 190 010

Date of Birth: 1-8-1935 SRINAGAR (Kashmir)

Educational Qualification:
-MBBS (MGM Medical College, Indore, M.P. 1958
-Diploma Otorhinolaryngology (King George's Medical College, Lucknow 1965)
-M.S. ENT (King George's Medical College Lucknow 1967)
-Diploma in Medical Audiology (DMA) Vellore
-FICS awarded in 1973
-WHO Fellowship in 1976

Posts Held:
-Professor and Head ENT department Govt. Medical College Srinagar since 1973
-Chief of Speech and Rehabilitation Centre of the State of J&K since 1970
-Retired as Prof. & Head ENT Deptt. G.M.C. Srinagar-1992

Academic and Other Activities
-Awarded S.T. visiting Fellowship by ICMR Delhi (India) in 1969 and 1970.
-Published more than 65 scientific papers in national and international journals.
-Conducted research projects on
i) Epidemology of cancer oesophagus in Kashmir.
ii) Incidence of ENT problems in school children.
iii) Incidence of deafness in new born infants in Govt. Hospitals.
-**Author of Text Book of E.N.T.** which is now read in India and abroad.
-Published pamphlets in Urdu and Kashmiri for training of Deaf and Dumb.
-Established hearing and speech centre and Neurootology clinic in S.M.H.S. Hospital Srinagar 1970.
-Organised annual conference of Association of Otolaryngologists of India in May 1969.
-Established the J&K State branch of Otolaryngologists of India and held annual conferences of the same.
-Started postgraduation in E.N.T. and got it recognised by MCI.
-Conducted an advanced operative course in Microsurgery for Ear and Parotid Gland 1989.
-Invited by King Abdul Aziz University Hospital Riyadh (S. Arabia) in 1994 to help them in ENT post-graduate Fellowship programme 1994.
-Visiting professor Jehlum Valley College in 1993 and 1995.
-Undergraduate and postgraduate examiner in ENT for many universities including Kashmir University, Delhi University, Banaras Hindu University Varanasi, Aligarh Muslim University, P.G.I. Chandigarh, Medical College Simla, Nag Arjuna University S.I.
-Senior selection committee member of U.P.S.C. of India; P.G.I. Chandigarh and Aligarh Muslim University.
-Member national deafness prevention committee.

Membership:
Life Member,
-Association of Otolaryngologist of India.
-Neuro-Otological and Equilibrometric Society of India.
-Indian Speech and Hearing Association.
-International College of Surgeons.
Twice elected as president of Medical College Teaching Association.

From the editor
Prof. Mir Maqbool is a dedicated, noble, hard working, disciplined, intelligent, God fearing doctor, teacher, clinician, social worker and a preacher of humanity and education. He is trying his best to promote basic education to the people of valley. His students, colleagues and patients respect him a lot.

Meet The Professor

Professor Upendra Kaul
M.D, D.M, FACC

JK Practitioner 1996; 3:2 122

Date of Birth: 14-6-1948 SRINAGAR (Kashmir)

Permanent Address:
N 221 Greater Kailash I, New Delhi - 110 029

Academic Qualifications
- 1970 MBBS, Delhi University
- 1975 MD (Medicine) Delhi University
- 1978 DM (Cardiology) Delhi University
- 1984 Post Graduate Training in Cardiology (Australia Post Graduate Federation of Medicine)
- 1988 Membership of National Academy of Medical Sciences
- 1995 Fellowship of American College of Cardiology

Experience
1975 to date : Worked as Lecturer in G.B. Pant Hospital; Assistant Professor, PGI - Chandigarh. Currently Professor, Cardiology, All India Institute of Medical Sciences, New Delhi.

Areas of Special Interest
Interventional Cardiology; Electrophysiology, Catheter Ablation, Cardiac Pacing; Surgical Cardiology and Acute Coronary Care.

Visiting Professorship
Royal Melbourne Hospital, Victoria, Australia; Cedars Sinai Medical Centre, Los Angeles, USA; University of Birmingham at Alabama, USA; and Washington Adventist Hospital, Takoma Park, MD, USA.

Membership of Professional Organisations
Vice President : Cardiological Society of India, 1990-1991 and CSI, Delhi Branch, 1993-1995

Editorial Adviser
Indian Heart Journal - Pacing and Electrophysiology - 1985, 1987, 1988, 1990. Coronary Artery Disease - 1989, Interventional Cardiology, 1990, 1991

Chief Co-ordinator - National PTCA Registry of India, 1991-1993 Convener Interventional Council Cardiological Society of India.

Awards

1969	Silver Medal for standing First in Forensic Medicine
1970	DR Thapar Gold Medal for being the Best Student in General Surgery
1983	Medtronic Award for the Best Scientific Paper
1986	Searle Award by the Cardiological Society of India
1987	Shakuntala Amirchand Prize by the Indian Council of Medical Research
1988	Awarded Membership of National Academy of Medical Sciences
1989	Awarded Dr. DK Paul Chaudhry Oration Award of the Indian Medical Association
1991	Awarded Uma Rani Banerjee Oration of the Cardiological Society of India
1993	National Press India Award for meritorious services in the field of Medicine
1994	Dr. Deshpandey Memorial Oration CSI - Vidharba Chapter, Nagpur

Publications
More than 325 publications in National and International Journals.

Number of guest lectures given during international meetings on Cardiology, Pacing, Angioplasty etc., held in USA, USSR, France, China, Indonesia, Singapore, Australia, Netherland, etc. from 1985-1995.

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In Memorium

Late Dr. Abdul Ahad Guroo

(1949-1993)

JK Practitioner 1996; 3:2 123

Dr. A.A. Guroo was born at Doabgah (Sopore) Kashmir on April 13, 1940 in a business family of repute. After his early education in Sopore he passed MBBS from Banglore (India) in 1964. He passed M.S. (Surgery) from Lucknow (India) in 1969. In 1978 he passed **M.Ch.** in Cardiothoracic Surgery from All India Institute of Medical Sciences Delhi. In 1980 he underwent fellowship in CVS in USA. After training from AIIMS he joined back to SMHS Hospital Medical College Srinagar as Asst. Professor Surgery till 1982. He joined Institute of Medical Science Soura, Srinagar (Kashmir) as Associate Professor of CVS in 1982 and headed the department as **Professor and Chairman** from 1984 to 1993.

Prof. Guroo started the open heart surgery at the Medical Institute, Srinagar in 1984 and through his untiring efforts and selfless dedication brought the department to the international level. He was always encouraging and helpful to his colleagues and a source of inspiration to the young medicos. To his patients he was no less than a Masiah.

Besides an excellent surgeon, humanitarian, social worker, he assumed the role of a freedom fighter. He started the Jehlum Valley Medical College in Srinagar, first of its kind in the state.

After a hectic week at the institute he would not rest even on sundays which he reserved for the poor and needy, patients of the native area whom he would treat free of charges. He was **assassinated on 31st March 1993.** He was burried at Idahgah (Grave-yard) Srinagar. In his martydom we have lost the greatest son of Kashmir and the medical profession has suffered an irrepairable loss. May his soul rest in peace Ameen.

Written up by:-
Prof. Ab. Ahad MS.
Head Deptt. of ENT, GMC Srinagar.

I have lost a great friend, colleague and a great surgeon of international repute.

May God give peace to his soul..

From Prof. Gh. Rasool Mufti
FRCS, Consultant Urologist (U.K)

I knew Late Prof. Abdul Ahad Guru for more than 15 years and had the privilege working with him for a long period. He was the founder of Thoracic Surgery in the valley of Kashmir. I found him a dedicated, bold, courageous and competent Surgeon of par excellence. He had the immense desire of bringing the department of Thoracic Surgery SKIMS to the national standards for which he always got best co-operation of his colleagues both at SKIMS and SMHS Hospital.

It was most painful and unfortunate for the community that he was martyed at the peak of his career.

"May God bless his soul"

Dr. Ashaq Hussain Naqashbandi (M.S;MCh)
Associate Professor & Head CVT Division
Deptt. of Surgery SMHS Hospital Srinagar.

What I know about this Healer of Heart :-

He was really a humourous, courageous surgeon and human being. During his last years of life, he spent few months as visiting professor at RIYADH and then in 1992 performed Haj along with his family. I found him always worried, restless to return back and to serve his countrymen. His death was mourned throughout the state as well as abroad. He will be always remembered by his students, friends, patients and colleagues. May God give peace to his soul.

G.M. Malik M.D; FACG
(Editor)

"Life As a Medical Student In The United Kingdom"

JK Practitioner 1996; 3:2 124-125

On the day I received the confirmation letter from the University of Wales, College of Medicine (UWCM) for a place in the first year. I felt as if I had been given the passport to fulfill my maim ambition in life - to become a doctor. My wish to study medicine had been not just a desire but an ambition. Most student enter medicine after completing their A-levels at the age of 18 years. I did not , as I was unsuccessful in entering medicine after my A-levels. This did not discourage me, but it gave me additional incentive to work harder. I did a B. Sc (Hons) degree in genetics after my A-level and I was successful in entering medicine following my degree. As a mature student at the age of 21 years, I was more confident in class discussion and tutorials than the post-A-level students.

In the first two years of medicine, we were taught anatomy, physiology, and biochemistry in great deal. The emphasis in the first year was on acquiring a firm understanding of the basic principles of these three subjects. The second year was much more interesting as the teaching eventually got down to the topics of clinical relevance. In the second year, the physiology and biochemistry tutorials were particularly stimulating as we were given the opportunity to present topic of interest in our tutorials and discuss current research findings with our tutors. This led to greater use of the library as a source of information. My physiology tutor was a neurophysiologist, and I took every opportunity to learn more from him about neurotransmitter involvement in neurological disease. Also, I presented a talk on "cerebrovascular accidents: before my physiology tutorial group. The physiology and biochemistry practicals were of little benefit in aiding our understanding of the subject. The practical schedule contained too much detail on the experiment itself and not enough guidance for us to make an inference based on our results.

The anatomy course in the pre-clinical years was excessively, and unnecessarily, detailed. In the first year, we studied the gross anatomy, histology and embryology of the upper and lower limbs, the thorax and abdomen, and the pelvis and perineum. Gross anatomy included nine hours of dissection every week, an activity which we never undertake again. In the second year, we studied the anatomy of the head, neck and back. We had lectures and practicals on neuroanatomy in the second year. These were very interesting and of great relevance in understanding neurophysiology and neurological disorders.

In the first year, sociology was our compulsory subject. The topics covered in sociology were all medically-related; for example. AIDS, the doctor-patient relationship, and drug addiction amongst many others. Psychology was our fourth compulsory subject in the second year. In my opinion, this subject is as important as the other three, as much of the morbidity in the community these days is due to psychosomatic illness.

At the end of our first year, we had internal exams in the four subjects studied in the first year. We were under more pressure in our second year as we had our 2nd MB degree exams at the end of this year. These exams were not too traumatic for those who had been studying conscientiously throughout the two years and doing well in the termly assessments. Anatomy and physiology revision took up sixty per cent of my revision time as we had such vast amounts of information to learn for these two subjects. Biochemistry and psychology were not as much of a pressure and took up only forty per cent of my revision time. I was fortunate enough to be awarded four distinctions in my 2nd MB exams.

The pre-clinical years introduced most students to the art of self-discipline. The majority of us had enough common sense to realise that the only way to get through the first two years of medicine (successfully!) was to spend numerous hours memorising everything that we were taught. In order to preserve our sanity during these two years; we put into practice the saying. "there is a time to work and a time to play", and when medical students "play" they do it with a hundred percent enthusiasm. For most medical students, socialising implies going to the medical students club (Medclub) on a Friday evening, visiting a night-club over the weekend, or simply joining some friends for a few pints of beer at the local public house. There are alternative social activities for those who are teetotallers, such as myself. My social life includes going with friends to sample the cuisine at restaurant in this area, visiting the cinema, or preparing dinner for my friends and entertaining them with my Urdu shayari (poetry) afterwards.

The third year, our clinical year, which is what I am in at present, is very different to the pre-clinical years. This is partly because we are isolated from the hospital in the first two years, with the only clinical input being a three hours clinical teaching session at the hospital on Wednesday mornings. We begin our third year with an introductory two week in which we are introduced to the hospital environment and taught basic medical procedures such as taking blood, venous cannulation, and emergency aid. The next 15 weeks of the third year are spent on two medical firms and one surgical firm being of 5 week duration. Most students are posted at least at two different hospitals during these 15 weeks. During this introductory period, all students are attached to a neurology firm for one week. In the last term of the third year, students are attached to a medical firm for 5 weeks and a surgical firm for 5 weeks. A clinical competence exam is taken at the end of each of these attachments. During the third year, all students have a 5 week attachment in dermatology and a week attachment in hematology.

Lectures in the first term of the third year are based on topics relevant to medicine or surgery, these are given only on Monday mornings. The remainder of the week is spent clerking patients on ward or in emergency admissions, attending out-patient clinics, or assisting surgeons in the operating theater if posted on a surgical firm. In the second term of the third year, we begin our teaching in pathology, pharmacology and community medicine. We are also given lectures on medical statistics. these subjects are examined at the end of the third year, together with written and clinical competence exams in the medicine and surgery.

During the third year, all students undertake a child development project and a cancer project. The former involves following the physical and psychosocial development of an infant from the time of its birth until its first birthday. The cancer project requires the student to spent time with a patient in the terminal stages of cancer in order to learn more about the disease and its psychological impact on patients and their relatives. Both of these projects are exercises in self directed learning.

Students acquire many new skills during the third year, the most important is being able to communicate with patients in a manner which gains the patients confidence. The first time that a patient communicates their medical and personal problems to you, a total stranger, and allows you to examine them, you begin to feel responsible for that person's welfare. Talking to patients every day teaches you more about the realities of life, and even those of us who are good at hiding behind textbooks have to come out into the open.

Our second set of degree exams are taken at the end of the first term of the fourth year. The subjects examined are pathology, pharmacology and community medicine. These exams are thought to be harder than the 2nd MB exam, but I cannot comment on this as yet, as these are a part of my future and not my past.

After passing the 4th MB exams, fourth year students begin their block attachments in the medical specialities of cardiology, anesthetics and intensive care medicine, child health, and, orthopedics, geriatric medicine, psychological medicine, obstetrics and gynecology, radiology, dermatology and general practice.

At the start of their final year, students spend a two months elective period working in a medical speciality of their own choice. Most students prefer to spend these two months working abroad in a country of their choosing. The remainder of the final year is spent getting more experience in medical and surgical firms before taking the final degree exams at he end of the year.

Medicine, for me, is not just a degree but the fulfillment of an ambition. In my opinion, for students to do well in medicine they need to have an eagerness to learn as much as possible from doctors and patients alike. In order to get the most out of the medical course, you have to enjoy every minute of it. If I had a second chance to choose a career. I would not hesitate in choosing medicine.

Author :
Monica Mathur (3rd Year Student)
University of Wales, College of Medicine,
Heath Park, Cardiff, CF4 4XW. (U.K)

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Issues Faced by IMGS and Programme Directors in USA

JK Practitioner 1996; 3:2 126-127

A) Special Issues Facing International Medical Graduates

Most IMGs who come to United States for training are in their 20s. Many are entering the United States for the first time, and the challenges begin at the airport. Questions, such as arrangements for pickup, accommodations, banking, Social Security must be addressed. First impressions for IMGs are long lasting.

Abbreviations and Cultural Differences

As an intern, I remember seeing a hypertensive patient with chest pain who was admitted as R/Ọ, MI, CAD, ASHD, HCVD. The usage of commonly understood abbreviations in the United States can be quite intimidating for an IMG who is unfamiliar with them. A year later, I recall being called to the emergency room at Queens Hospital Center in New York City to see a young man with "SS crisis". Having never seen a black patient, let alone one with sickle cell disease, I had to quickly refer to ***Harrison's Principles of Internal Medicine*** and learn about hydration, analgesia, and TLC for this condition.

Similarly, imagine a resident's first experience with a tremulous, agitated, febrile patient who insists that he drank with you in a bar the previous night and who sees elephants on the wall. A resident's first experience treating alcohol-withdrawal syndrome can become a life-long memory, particularly if he or she hails from an area where alcoholism simply does not exist.

The IMG is expected to treat disease and situations that he or she has not previously encountered. Thus, it is important for program director and coordinators to be patient and understanding of the IMG's special needs.

Interaction with Nurses

In the British system, a ward nurse is referred to as "Sister". Imagine the reaction of an IMG when the head nurse, in no uncertain term. says "Honey. don't call me sister; you and I know we're not related. We may develop a relationship, but not as brother and sister." I had just such experience. The IMG needs patient counseling about the relationships among members of the health care team.

Other Issues for New International Medical Graduates

Invariably, my house officer tells me they are overwhelmed by paperwork and regulatory concerns, IPRO. diagnosis-related groups, length of stay, term that are unfamiliar to IMGs. Most IMG trainees believe that these regulations detract from delivery of good patient care. They also feel that physicians in the United States are overly dependent on "laboratory medicine." with not enough emphasis on bedside medicine. Most IMGs come for medical systems where "high touch" rather than "high tech" is emphasized.

B) Issues for Program Directors

Most program directors face this question: Where do IMGs come from and how can we best evaluate them?

Worldwide, the number of medical schools increased from 646 in 1955 to 1,351 in 1985. It is no surprise that the caliber of medical schools varies widely. The top 10 "exporters" of IMGs to the United States are India (20%); Pakistan (12%); the Philippines (9%); Russia (3%); Egypt (2.6%); the Dominican Republic, Syria, and the United Kingdom (with 2.5% each); Germany (2.3%); and Mexico (1.8%). How does one assess graduates from schools where English is not the mode of instruction? It is difficult to evaluate candidates and credentials from schools in non-English-speaking countries.

When we compare the status of medical schools in the United States in 1910 (6) to the present medical system in other nations, several parallels emerge. Many propriety schools, "diploma mills," have emerged. It is the program director's challenge to determine the genuinely high-caliber schools and refrain from the common tendency to group all IMGs into one category. Several parallels exist between Flexner's predictions in 1910 and the status of IMGs in the 1990s.

After reviewing New York's 11 medical schools, Flexner made the following recommendations:

New York. 1910: General Considerations - Population 8.7 million. 14,117 Physicians - Ratio 1:617, Schools 11, PG:4.

New York has double duty and opportunity in medical education. Population is well distributed; communication is easy; roads are good; education facilities abundant and doctors superabundant. New York must be asked to do more than produce doctors. Its vast hospital and university resources should make it the Berlin or Vienna of the continent, a genuinely productive contributor to medical progress; the center to which physicians will repair to freshen their knowledge and to renew their professional youth, to which the young graduate from the interior Pittsburg, Ann Arbor, Madison, Iowa City - will look for the extension of his scientific and clinical experience (Flexner Report:275).

Flexner's recommendations from 1910 appear to be fulfilled. The United States is the center for advanced medical education in the world. States such as New York have played a preeminent role in this, now with its 13 medical schools, graduation of 10 % of all U.S. medical students. and training of 15% of all physicians in advanced medical specialities. The only change from Flexner's view is that many trainees in New York State are not coming from the interior, from Pittsburgh, Ann Arbor. or Iowa, but from the exterior-India, Pakistan. the Philippines, Kashmir, Egypt, Russia, the United Kingdom, the Dominican Republic, and so on. Flexner would be pleased, nonetheless, to see that the United States has achieved worldwide recognition and stature in advanced medical education.

Faroque A. Khan, MB, FRCP(C)
Chairman Department of Internal
Medicine Nassau County Medical Center
East Meadow, NY
(Author of Book on Foreign Medical Graduates
Published by American College of Physicians
U.S.A)

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Communications to Editor

Dear Dr. Malik,

Thank you very much for your letter dated 26 August 1995 and the enclosed complimentary copy of the Journal you edit. I was indeed impressed by the quality of the articles in your journal reflecting no doubt on your commendable editorship. It was a pleasure for me to write an editorial on Coronary Heart Disease in Asians Living Abroad and I want to assure you that if the need arises in the future I would be willing to contribute to your journal again. Thank you very much and looking forward to the other issue.

Dr. LDKE Premawardhana
Consultant Physician
Rhymney Valley Health Unit
Caerphilly District Miners Hospital
St. Marins Road, Caerphilly,
Mid Glamorgan, CF8 2WW (U.K)

Dear Dr. Malik,

Thank you for sending a copy of the pracitioner (International). I was pleased to see the very interesting standard articles in this Journal. I am sure this is going to be of tremendous help to local doctors and the community as a whole. Hopefully in future I should be able to send you papers for possible publication in your Journal.

Dr. M.R. Teli M.D, MRCP
Consultant Physician & Gastroenterology,
Louth & District Health Care,
County Hospital High Holme Road, Louth
Lincolnshire LN 11 OEU, U.K

Dear Dr. Malik,

It is more than two months now I have striving to adjust to the strange environment of Europe. Providing a hi-tech environment it takes time to familiarise to the change. I am sure the Journal (JK Practitioner) will touch new heights under your able stewardship. Your academic brilliance will go a long way to explore new horizons in the field of Medical Sciences. With the introduction of Professionalism the journal will achieve the national comparable standards and that area needs your attention. The quality of papers and printing (get up) needs to be improved.

Wishing you best of everything in life.

Dr. Syed Amin Tabish
MAMS, FIMSA, FRIPH (LON)
University of Bristol, Department of
Health (Social Medicine, Canynge Hall,
White Ladies Road, Bristol BS8 2PR U.K

Dear Dr. Malik,

Thank you very much for a copy of the November edition of JK Practitioner. The layout is vastly improved and the front page looks excellent now. I should like to congratulate you and those of your colleagues who are responsible in bringing about such a transformation in this Journal. The contents are very revelant to topical issues. I showed the journal to some of my colleagues who were full of praise for your efforts.

In connection with my earlier suggestion, I am enclosing an article written by a medical student at my request. She has chronicled her activities as a medical student, reflected on medcal education in this country and on some of the social activities which medical students usually get into. I hope you will find it of interest and will feel that it is suitable for publication in your journal. I think many of the doctors and medical students there would find it interesting. This article could feature as an occasional overseas diary or as a regular feature. You could approach some of your friends in the states and elsewhere to send you similar submissions. We will try and generate some more interest and hopefully you may get further submissions of medical and general interest. We will do all we can do to help you establish.

Dr. M.A. Mir, MRCP
Senior Lecturer & Consultant Physician
Department of Medicine, University Hospital
of Wales, Health Park Cardiff CF 4 4XW, U.K

Dear Dr. Malik,

It was unfortunate that I could not meet you at Srinagar and further more I am really sorry, that it took me so long to answer your letter. I was impressed with the fact that such a service as Practitioner is being provided in Kashmir. It is an excellent piece of work and I must congratulate you for it. What kind of articles would you like me to provide ?

Write me about it. I can contribute about modern drug development. I have been associated with the drug development for the last 38 years and it has been a fascinating journey. Meanwhile, I am enclosing some information about my recent activities.

I wish you a Happy New Year and all the luck in the world with enterprise. Keep up the good work.

Dr. Ghulam Nabi Mir
President & CEO, DMR Laboratories
P.O. Box 394, Buckingham, PA 18912, U.S.A

Dear Dr. Malik,

Thank you for your letter in which you requested some articles for the Medical Journal "JK Practitioner". We are in the process of drafting several manuscripts. As soon as we have finalized my most recent work in Rotablation I will be happy to share a version with you for the JK Practitioner.

Dr. Fayaz A. Shawl, MD, FACC
Director, Interventional Cardiology
Washington Adventist Hospital
Clinical Associate Prof. of Medicine
(Cardiology) George Washington
University Hospital, U.S.A

Dear Dr. Malik,

I received your letter dated 4th Jan. 1996, this morning. Please find a copy of my article: Islamic view on Organ Transplantation. Please also find articles on Islamic teachings and Cancer Prevention Part: Tobacoo and Cancer Part I: Alcohol and Cancer. Please let me know, if you agree on publication and send me a copy of your Journal.

Dr. M. Albar, FRCP
Consultant of Islamic Medicine,
King Fahad Medical Research Center
King Abdul Aziz University Jeddah (K.S.A)

Dear Dr. Malik,

Thank you very much for sending to me the January-March 1996 issue of JK Practitioner in which my article on "Current concepts in the Management of Gallstones disease" is published. The issue has come out very well. This has been possible only because of your continued efforts. Congratulations!

With best wishes and warm personal regards,

Sincerely,
Rakesh K. Tandon, M.D, P.hd
Head Gastroentrology & Nutrition Deptt.
AIIMS, New Delhi

Dear Dr. Malik,

Thank you for your letter regarding abstracting information from the American Journal of Gastroenterology for your Journal J.K. Practitioner. I have discussed this with our Editorial Board and they are in agreement in allowing you to provide brief abstracts of our publication. Thank you for getting in touch with me.

Rowen K. Zetterman, MD, FACG, FACP
Editor In Chief, American Journal
of Gastroenterology & Professor of
Internal Medicine Hepatic Transplantation
University of Nebraska Medical Center
600 S, 42nd Street, Omaha, (U.S.A)

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Yours sincerely
Saleh Al Deeb MD, FRCP(G), FRCP(Ed)
Editor Saudi Medical Journal
Armed Forces Hospital, P.O. Box 7897,
Riyadh 11159, Saudi Arabia.

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Editorials:

-Tobacco Smoking, The Health Hazard & Beyond 128 Sheikh Aejaz Aziz
-Brain Death 129 A. Salam, A. Wahid, M.A. Akhter

Guest Article:

-Management of Chronic Hepatitis 131 M.P. Sharma

Clinical Rewiew:

-Post-MI Mechanical Complications 136 P.K. Shah, J.S. Goodman
-Biochemical Assessment of Hepatobiliary Disorders: Traditional Approaches with New Strategies 142 M.M. Mir, A.R. Khan, Z.A. Shah

Origional Articles:

-A Survey of the Value of Ultrasound as a Predictor of the Findings at ERCP in a District General Hospital 145 M.R. Teli, I. Gobden
-Chemical Composition of Gall Stones 151 A.M. Bhat, I.A. Mir, O.J. Shad, N.A. Wani
-Role of Colposcopy in Cervical Cancer Detection 155 W. Khannam, S. Hamidi, A. Rashid

Case Reports:

-Survival in Water Intoxication 157 S. Banzal, F.A. Bhat, H.S. Rissam, Jamal A.
-Carcinoma of the External Auditory Meatus 160 N.A. Bhat
-Kartagener's Syndrome with Simple Hepatic Cyst 162 S.A. Kadla, S.A. Khan, M. Agaz, M.A. Wani, T. Hussain, S. Rashid, J. Basu, G.M. Malik
-"Xeroderma Pigmentosa & Skin Cancers" 165 S.A. Ahad, M. Latif, Zahoor A., Naseema C.
-Supraglottitis During Pregnancy 167 Parveez A. Mir

Short Case Report with Clinical Review:

-Extreme Metabolic Alkalosis & its Management: A Review of Two Cases 169 R. Bashir, T. Mir, F. Khan

Special Articles:

-Doctor & Patient Relationship "Holding a Hand is Often as Important as Examining One" 173 Lawrence H. Brandt
-"Islamic Teachings & Cancer Prevention" Tobacco & Cancer 178 M.A. Albar

Imaging Today:

-Ultrasonography in Chest Medicine 185 G.Q. Khan, Iftikar A.
-Magnetic Resonance Cholangiography- a Threat to Diagnostic ERCP? 187

Practitioners Column:

-Laboratory Diagnosis of HIV Infection 188 G. H. Hajini, S.T. Hussain

Students Page:

-"The Psychology of Resuscitation" 193 Ralph M. Shenefelt
Clinical Images: 194 Mir N. Ahmad

Hospitals Today:

-Implementing & Sustaining Health Service Quality 195 Syed Amin Tabish

Overseas diary:

-Future Prospects for International Medical Graduates in the United States 198 Tanveer P. Mir, F.A. Khan

Meet the Professor:

-Prof. Brij Mohan Bhan 201 M.S. Misgar
-Prof. Mohd. Aslam 203
Obituary: 204

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- -> **Sepsis Syndrome Therapy**
- -> **Application of Laser in Surgery**
- -> **The Chromide of Growth Harmone Insensitivity Syndrome**
- -> **Migraine - New Horizon**
- -> **Lithium & Kidney, Trace Elements in Head & Neck Cancer**
- -> **Meet Professor - S.Z. Ahmad (Ex. Director SKIMS/Ex. PMC Medical College Srinagar)**
- -> **Overseas Diary - MRCP Examination by Dr. M.A. Mir**
- -> **Cancer Prevention & Alcohol**

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Tobacco Smoking, The Health Hazard and Beyond

JK Practitioner 1996: 3:3 128

Global tobacco consumption exceeds 7 million tones, costing $200 billions per year. With 600 billion cigarettes being smoked every year, there are 1.1 billion smokers world-wide, 41% being from the developing countries and there are 8% women smoking regularly. Developing countries account for 3/4ths of the world population, the extent of cigarette smoking per adult consumption rose from 1150 per year to 1400 and is still increasing at the rate of 1.7% per year. In the developed world, the cigarette consumption has dropped from 2800 cigarettes per adult in early 80's to 2400 in the early 90's. There are about 3 million deaths reported due to smoking with 2/3 rds of the deaths occuring in the developing world. The death toll is expected to rise to 10 million per year by 2020, with 70% of these occuring in the developing world (WHO).

In India, Tobacco was introduced only in the 17th century by the Portugeese, but within a short period, its use spread all over the country. Currently, nearly half a million hectares of the cultivable land is used for growing 5,00,000 tonnes of tobacco annually contributing over Rs. 1550 crores to the state exchequer, besides employment. The National level survey indicates tobacco usage prevalence of 35% in men and 12% in women. Data collected by the National cancer registry programme of ICMR indicate that nearly 1/3rd of cancers occuring in India are attributable to the tobacco use, and mortality rates in smokers is nearly 1.4 to 1.6 times higher than the non-users. There are approximately 800.00 deaths annually attritubale to the tobacco use in India.

Tobacco harms environment besides causing a burning hole in the pocket. In some tobacco producing countries, people have to travel over 10 Kms just to find wood for the domestic use. Growing tobacco involves extensive use of fertilizers and pesticides producing fiscal dent. Women and children working in tobacco industry face nicotine poisoning. Environmental tobacco smoking is significant in adversely contributing to the tobacco related health hazards. There is hardly any biological activity in human that is not effected badly by tobacco use.

Tobacco induced long term self-destructive behaviour - the chronic suicide, complemented by the fossilization of the spiritual beliefs and value systems has compounded the nullity of what Dostoevsky defined as "tomorrow's zero". This mutated life-style simply represents drudgery and tedium of millions. An honest socio-political will and health education seems mandatory to create a smoke-free environ today.

Sheikh Aejaz Aziz
MD; DM (Oncology)
Consultant SKIMS.
Srinagar. Kashmir-190001
India.

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Smoking Hazards (1996)

GENEVA: The World Health Organisation (WHO), recently warned that the number of people who die as a result of smoking may surge to ten million by the year 2020 from the present three million victims annually unless tobacco consumption was curtailed drastically.

The WHO in a report, issued ahead of May 29, 1996 the global no smoking day, said tobacco-related illnesses claimed one life every ten seconds on an average globally.

Brain Death

JK Practitioner 1996; 3:3 129-130

Brain death describes the condition in which all functional brain activity is permanently lost due to irreversible structural damage to the organ. As with irreversible asystole, the condition is absolute and is both medically and legally accepted as defining death of the person in many countries world wide[1]. The diagnosis of brain death has assumed increasing importance in recent years, firstly because of the increasing difficulty of deciding in patients with brain damage whether it is justifiable to maintain life indefinitely with assisted respiration and other supportive measures in intensive care units (difficult resource allocation) and secondly because of the difficult question of deciding when it may be concluded that the cerebral lesion is irreversible, that cardiac arrest is imminent and that preparations may be made to remove viable organs for the subsequent transplantation[2].

Brain death results from total cessation of cerebral blood flow and global infarction of the brain at a time when cardiovascular and respiratory functions continue of its own or preserved with artificial support. It is the only type of irrevocable loss of brain function currently recognized by law as death. It may take a week's time in patients of brain death for cessation of cardiovascular functions even if full medical support is omitted or longer with full artificial support, hence emphasis the expensive and inhumane futility as such efforts. Making the diagnosis before cardiac decompensation and arrest occurs, provides the highest quality of organs for transplantation. Organ transplantations from brain dead patients are very common in most of the industrialised countries, even now all over Muslim countries[3].

Pathology of brain death (respiratory brain):

There is a widespread necrosis and edema without inflammatory reaction (liquifactive necrosis) transtentorial (temporal lobe) and cerebellar tonsillar herniation. Brain swelling causes increased intracranial pressure and an absence of cerebral blood flow.

Diagnosis:

The President's Commission for Study of Ethical Problems in Medicine and Biomedical and Behavioural Research of USA has set forth the criteria for the diagnosis of brain death. They have been adopted specifically or in principle in all parts of USA with no more than minor changes. They closely resemble principles or statutes adopted internationally[4].

Clinical criteria:

1. Cerebral functions are absent. There must be deep coma with no detectable cranial, behavioural or reflex responses to internal or external stimuli
2. Brainstem functions are absent. There must be no evidence of spontaneous or reflex activity received by or emanating from any brain structure lying rostral to foramen magnum
    - a) Cranial stimulation above the 2ndcervical dermatome elicits no cranial or bodily response, conversely, no stimulation at or below the cervical level elicits any cranial functions.
    - b) The following must be specifically absent: corneal-pupillary and somatic facial reflexes, caloric oculovestibular reflexes and oropharyngeal reflexes[1,5].
    - c) Sustained apnea must exist despite adequate endogenous hypercarbic ($PaCo_2$ 60mm Hg) stimulation.

3. Coma must be due to a known irreversible cause:
    - a) Severe cases of head trauma, spontaneous intracranial haemorrhage, intracranial infections, cerebral global anoxia, ischaemia, hyperinsulinism, hepatic encephalopathy etc.
    - b) Drug intoxication must be considered & ruled out in every case of deep coma particularly when the precipitating event is unknown, therapeutic coma (barbiturate coma) or neuro-muscular blockade has been employed. When in doubt one must wait.
    - c) Deep hypothermia must be corrected. Unless EKG asystole is found it is wise to warm cold bodies to 33°C or more, especially when sedatives or alcohol has been given or ingested.

4. An adequate time must be allowed to assure that clinical signs do not change. In USA 12 hours' interval between first and second observations in adults and 24 hours in children of 1-16 years' age is given. In instances where organ transplant is contemplated or other conditions justifying shortening the interval of observations, it can be reduced to 6 hours in

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adults and 12 hours in children if one of the following objectives measure obtained.

a) A technically satisfactory EEG shows electrocerebral silence (hypothermia or drug intoxication are specifically ruled out) or

b) A technically satisfactory test shows no brain circulation (radionucltide brain scanning, cerebral angiography and transcranial doppler[6]).

It is often advisable to delay clinical testing for upto 24 hours if cardiac arrest has caused brain death.

Pit-falls and special considerations:

For children between 7 days and 2 months, 2 examinations and 2 EEG's, 48 hours part are required[7,9].

Spinal reflex activity after bain death. Deep tendon reflexes of truck and extremities commonly occur in brain dead bodies as do extensor planter responses. Complex spinally engendered reflex movements may occur during final apneic tests[8].

Physician's responsibility:

The psychosocial implications of death are always difficult for both the physician and patients's family. The difficulty is compounded in patients in whom there are superficial signs of survival.

a) Once the diagnosis has been determined, the physician should explain the meaning of the syndrome carefully to the patient's family.

b) The clinical record must reflect the decision making process and should document the signs carrefully. Two physicians, neither of whom is a member of the transplantation team should agree on the diagnosis in the record.

Abdul Salam, MD., DM Neurologist,
A. Wahid, MD Asstt. Prof.
M.A. Akhter, MD Professor
SKIMS, Sgr - Kashmir 190001

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Management of Chronic Hepatitis

M.P. Sharma*

JK Practitioner 1996; 3:3 131-135

INTRODUCTION

The last decade has seen tremendous advances in the knowledge of chronic hepatitis, with regard to its types, etiologies, diagnostics and treatment. Availability of ELISA based tests for antibodies to various fractions of the hepatitis B genome and to hepatitis C virus (structural and non structural antigens), detection of HBV-DNA and HCV-RNA by hybridisation assays or polymerase chain reaction (PCR) and detection of autoantibodies have made the diagnosis of chronic hepatitis more sensitive and specific and evaluation of response to therapeutic modalities more objective.

The terminology of chronic hepatities has been recently modified based on the recommendations of the International Working party constituted during the World Congress of Gastroenterology, held in 1994.(1) The 6 month period usually required as essential for the diagnosis of chronic hepatitis is now considered as intended for study of large cohorts and may not be essential for individual patients who may merit treatment earlier when all other features are present. Also, keeping in line with the earlier recommendation of the International Autoimmune Hepatitis Group, autoimmune hepatitis is considered a priori chronic, and does not need a 6 months period for its definition.[2]

CHRONIC HEPATITIS B

Evaluation:

The diagnosis of chronic hepatitis B requires the following criteria to be fulfilled - (i) evidence of liver disease for 6 months or more, usually in the form of elevated serum aminotransferases with presence of HBsAg, HBeAg or HBV-DNA (ii) Histological findings of portal inflammation, lymphocytic piecemeal necrosis, bridging or confluent (lobular) necrosis with or without cirrhosis. To distinguish chronic hepatitis B reliably from other types of viral hepatitis, stainable HBV antigen must be present (HBsAg or HBeAg).

Diagnosis:

Progressive chronic hepatitis B is believed to occur with persistence of viral replication (HBeAg positive, HBV-DNA positive), viral mutations or transient exacerbations in viral replication. The corner stone of diagnosis of chronic HBV infection is the detection of HBsAg; total anti HBc is present in almost every case of chronic HBV infection. [3] Igm-anti HBc is not detectable by the usual commercial assays, but is detectable by more sensitive assays; the 7-8S form of IgM-anti HBc predominates in chronic HBV as contrasted from the 19S form which predominates in acute HBV.[4]

In established chronic HBV infection, HBeAg tends to indicate active replication, and tends to disappear in about 50% patients over a period of time (seroconversion), this phenomenon being associated with a brief episode of elevation in serum ALT levels. Except in situations where a HBcAg negative mutant virus is present, conversion of HBeAg positive patients to HBeAg negative indicates that the virus has entered non-replicative phase.

Indications for treatment:

Patients of chronic HBV infection who are HBsAg positive and have HBeAg and/or HBV DNA in serum with elevated serum ALT (at least twice upper limit of normal) merit therapy.

Those who are negative for HBeAg and HBV-DNA in serum should not be treated with alpha-interferon. Similarly, those with normal transaminases do not require therapy.

Goals of Therapy in Chronic Hepatitis B:

The goals of treatment in Chronic hepatitis B can be divided into the following:-

a) Short term goals -
  i) suppression of HBV replication - loss of HBV DNA, HBeAg, conversion to Anti-HBe.
  ii) remission of liver disease - resolution of symptoms, normalisation of ALT, improvement of liver biopsy.

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*Profesor
Department of Gastroenterology
All India Institute of Medical Sciences
New Delhi 110 029

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b) **Long term goals -**
  - i) loss of HBsAg - considered as "cure".
  - ii) prevention of progression to cirrhosis or hepatocellular carcinoma.

Drug therapy for chronic Hepatitis B:

The major goal of therapy is to suppress or eliminate the virus permanently to achieve remission of liver disease. The therapeutic endpoint in all clinical trials has been the sustained elimination of circulating markers (such as HBeAg, HBV-DNA and HBV-DNA polymerase). Clearance of HBsAg is more difficult to achieve but the proportion of patients who lose their HBsAg tends to increase with time after completion of interferon therapy.

In the last decade, various drugs have been studied as potential treatment for chronic hepatitis B. Of these, the Food and Drug Administration (FDA) of USA has permitted only alpha interferon to be used in patients. The list of drugs tried includes the followings:-[5]

1) Antiviral agents -
  - a) Interferons - alpha, beta, gamma
  - b) Adenine arabinoside (Ara-A)
  - c) Adenine arabinoside monophosphate (Ara-AMP)
  - d) Acyclovir, Gancicyclovir
  - e) Dideoxyinosine (DDI)
  - f) Ribavarin

2) Immuno modulators -
  - a) Corticosteroids
  - b) Interleukin - 2
  - c) Tumor Necrosis Factor - alpha
  - d) Thymosin - alpha
  - e) GM - CSF
  - f) Glycerrorhyzin (SNM-C)

3) Nucleoside analogues -
  - a) Virazide
  - b) 2-carboxydeoxy guanosine

4) Combination therapy
  - a) IFN/ara-A, IFN / - AMP
  - b) Prednisone / IFN
  - c) Prednisolone / ara-A / IFN
  - d) Acyclovir / IFN
  - e) Levamisole / IFN

Interferons - types, actions

Interferons are molecules that were first identified when incubation of chick chorioallantoic membrane was carried out with heat inactivated influenza virus, & the replication of the virus "interfered: with. Soon it was recognised that interferons, besides possessing antiviral actions, also acted as anti tumor and cell regulator molecules. Three classes of interferons were identified - interferon alpha (IFN-α) produced by leucocytes, interferon beta (IFN-β) produced by fibroblasts and interferon - gamma (IFN-γ) produced by lymphocytes. IFN-γ is also called a type II interferon or immune interferon. All interferon proteins are 166 amino-acid long, whose formation is controlled by genes-the genes for IFN-α and β are located on chromosome 9 and that for IFN-γ on chromosome 12.

The interferons used in chronic hepatitis are recombinant, and commercially available preparations include interferon Alpha 2a (Roferon), Alpha 2b (Intron A), Alpha 2c (Berofer), Alpha nI (Wellferon), n2 (Alferon), and Gamma 1b (Actimmune). The chief biological actions of interferons are :

- a) antiviral actions
- b) Cell regulatory activities e.g antiproliferative, imunomodulatory, regulation of gene expression, etc.

Antiviral effects -

Interferons are potent antiviral agents, and act by protecting the cell from the effects of viral infection ("sparing effect") or by rendering the viral infection short. This antiviral effect lasts only for a period of several days and thereafter decays. At the cellular level, this is brought about by - induction of several enzyme systems, transcription of a Mx gene that inhibits the transcription of the viral genome, inhibition of early viral mRNA, inhibition of uptake, penetration and uncoating of viruses etc.

Antiproliferative & anti differentiation effects -

Interferons inhibit cellular multiplication, and can either suppress the differentiation of some cells or enhance others. They also have an inhibitory effect on lattice contraction of fibroblasts and thus reduce scarring and fibrosis.

Immune effects -

The various effects described include enhancement of expression of histocompatibility antigens and B - microglobulin on plasma membranes, induction of natural killer cell activity, activation of interleukin - 2 receptors, induction of B- cell immunoglobulin production.

Alpha - Interferon in HBV

Alpha-Interferon can be administered in a dose of 5 million units (MU) / day subcutaneously or 10-MU three times weekly for a period of 4 months. Responders show a loss of HBeAg and HBV DNA with normalisation of serum ALT levels. This occurs in around 30-50% of patients, and this response appears to be sustained, even when patients are observed for intervals of as long as 3-7 years.(6) Reactivation of infection occurs in approximately 5-10% of individuals who undergo HBeAg seroconversion, usually within one year of completion of therapy. HBsAg clearance occurs in 10-15% patients within the first year of treatment, and in one study continued to occur in upto 65% patients upto 4 years follow-up.(6) Although 80% of those who are HBsAg negative are also HBV - DNA negative in the serum, polymerase chain reaction can detect HBV-DNA in the liver.(8) Liver histology improves within 1 year of completion of therapy, and in some studies, continues to improve for upto 4 years of follow up

The predictors of response to interferon therapy include: i) high initial levels of serum aminotransferases, (ii) low initial levels of HBVDNA in serum (100 pg/ml), (iii) history of acute hepatitis (iv) short duration of disease, (v) female sex, vi) absence of other major medical conditions such as renal insufficiency, HIV infection or immunosupressive therapy.(8)

Side effects of interferon are usually dose - dependent and may occure "early" (fatigue, fever, chills, myalgia and arthralgia) or "late" (myelosupression, mood changes, autoantibodies, thyroid dysfunction). Patients on interferon therapy should have regular blood counts and clinical assessment for psychiatric manifestations.

Evaluation of 16 randomised trials of interferon therapy in chronic hepatitis B, enrolling 837 patients showed that HBeAg was lost in 33% of 12% (in controls). HBV DNA was lost in 37% (if 17% controls) and HBsAg was lost in only 7.8% (if 1.8% controls).[17] The National Institutes of Health reported that sustained loss of HBeAg over a 3-6 year follow up occurred in 65% patients. About 10 - 20% of patients who clear HBeAg ultimately lose HBV DNA, compared to 50 - 100% of patients who clear both HBeAg. The response in Indian patients treated with 3 MU thrice weekly has been shown to be good - 50% lost HBeAg or HBV DNA (If 4.8% controls) and 35% ser5oconverted to Anti - HBe (if 4.0% controls).[18]

The response in decompensated cirrhotics, children, innumosuppressed patients, patients on dialysis etc. is poor and variable.

Other drugs and combination therapy

Thymosin fraction 5, a thymus - derived polypeptide that regulates maturation of T cells and interferon production has shown no benefit after 6 months of treatment. Thymosin - alpha has shown 86% loss of HBV DNA after 6 months of treatment, in one study.[9]

Ribavarin, a systhetic nucleoside, which had earlier shown limited effects on AST levels, HBsAg titer and DNA polymerase activity, has shown a renewed promise. In a pilot study conducted at the National Institutes of Health, moderate reductions in HBV DNA and ALT levels were observed with a dose of 800-1200 mg/d for 6 months.

The use of short pulse of corticosteroid followed by withdrawal and then interferon has been compared with interferon alone in a US multicenter trial. Nearly identical rates of HBeAg seroconversion was oberved in the two groups, around 40%; however, patients with low pretherapy ALT levels (<100IU/L) responded more frequently to the combination regimen than the group that received interferon alone. Perrillo advocates a 6 - wk pretreatment with corticosteroids in patients with low ALT followed by interferon.[10]

CHRONIC HEPATITIS C

Chronic hepatitis C is an inflammatory disease of the liver caused by hepatitis C virus, lasting 6 months or more, with the potential to progress to cirrhosis or associated with cirrhosis.[1] Symptoms of liver disease may range from mild fatigability to overt cirrhosis. The diagnosis is made on the basis of detection of antibody to hepatitis C virus (anti-HCV), either by ELISA or by a second/third generation recombinant immuno blot assay (RIBA).

Evaluation of chronic hepatitis C

First generation ELISA, based on detection of antibodies to the C100-3, a fusion polypeptide antigen representing part of the NS4 region of the HCV genome, is clearly an inadequate assay due to its lack of sensitivity and a high false positive rate.[11]

Second generation ELISA, incorporate two extra HCV-derived recombinant proteins, c22c and c200, while the third generation ELISA incorporates a NS5 epitope. The **third generation RIBA 4** includes bands 5-1-1, c100-3, c22-3, c33-c and NS 5 polypeptide and a positive result is indicated by positive result is indicated by positivity to two or more antibodies, while positivity to one antibody is an indeterminate result.

The antibody tests to HCV have a high degree of correlation to the presence of HCV viremia. Direct detection of HCV-RNA by conventional nucleic acid hybridisation techniques is difficult. but HCV-RNA can be detected by reverse - transcriptase polymerase chain reaction (RT-PCR). selection of PCR primers from the 5 - region has resulted in a greatly increased sensitivity of the assay; similarly, use of nested PCR can detect as little as one molecule of cDNA.

HCV-RNA usually persists in patients with abnormal serum aminotransferses and anti-HCV; however, even those with normal ALT values can have HCV-RNA. Unlike HBV. emergence of mutant populations of HCV does not always correlate with peaks of ALT.

Liver biopsy is useful in the evaluation of a patient suspected to have HCV -chronic hepatitis, since varying grades of fibrosis and cirrhosis may be present at diagnosis. Certain biopsy features considered to be characteristic of HCV include denise portal lymphoid aggregates, bile duct damage, bile duct dysplasia nd macrovesicular fatty change. Lymphocytic piecemeal necrosis is relatively mild. The severity of cirrhosis depends upon the duration of infection and the degree of inflammatoru damage at onset. In one study of post transfusion hepatitis C. 20% patients had cirrhosis after 10 years, whereas 6-71% had cirrhosis after 20-25 years.[12]

Goals of treatment

Since hepatitis C is an insidiously progressive disease, with a 50%-80% risk of developing cirrhosis and a 20% chance of developing hepatocellular carcinoma. and the chances of spontaneous clearance of the virus are extremely low. the long term goal of any therapy should be reduction of the risk of histological progression of liver disease and suppression/eradication of the HCV.

Interferon therapy

Recombinant interferon alfa-2b (r-IFN-2b) is the only approved treatment for chronic hepatitis C which is thought to act by directly inhibiting replication of the HCV by various mechanisms.

The **pretreatment evaluation** includes serologic confirmation of the disease by anti-HCV. presence of serum ALT elevation and a lever biopsy compatible with the diagnosis. Interferon is **contraindicated** in hepatitis decompensation. significant cytopenia (platelets < 70,000. WBC < 3000). autoimmune disease including untreated thyroid disease and severe psychological disorders. **Initial treatment** is started with 3MU subcutaneouslu thrice weekly for 24 weeks; **monitoring** is by blood counts and ALT at 1.2.4 weeks and then at monthly intervals. TSH at 3 month intervals and HCV-RNA at the end of therapy. **Relapses** may be treated with repeated courses, prolonged (?life long) treatment at fixed lower doses or titrated doses.[13]

The response of HCV to interferon is dismally poor. The largest trial conducted in the US involving 166 patients showed normalisation of ALT in 38% patients compared to 4% controls with significant improvement in liver biopsy scores.[14] Thirteen randomised controlled trials of interferon in chronic hepatitis C using 1-10 MU thrice a week for 1 to 18 months showed that the ALT response ranged from 35-73%, histologic improvement in 60-70% and loss of HCB-RNA.[15] However, majority of the responders (50-80%) relapse within 6 months of completion of therapy, and the overall cure rate fails to exceed 20-25%

The response to interferon is also predictably related to HCV genotype - types 2 and 3 show the best response while type 1b responds poorly. Also the level of viremia is correlated to IFN response - low level viremia responds better. Those who have a marked nucleotide diversity in the hypervariable region-1 (HVR-1) have a poorer response to interferon.

AUTOIMMUNE HEPATITIS

Autoimmune hepatitis (AIH) is an unresolving, predominantly periportal hepatitis associated with hypergammaglobulinemia and tissue autoantibodies. which is responsive to immunosuppressive therapy. The **clinical diagnosis of AIH** requires the exclusion of alcoholic liver disease. exposure to hepatotrophic viruses (although type II AIH is commonly associated with HCV). hepatotoxic drugs. or evidence of genetic metabolic disorders (-antitrypsin deficiency. Wilson's disease or hemachromatosis).

Laboratory findings include an elevated bilirubin in value. raised aminotansferases. mildly elevated alkaline phoshatase and hyperglobulinemia with a >1.5 fold increase in serum IgG. Circulating antibodies include antinuclear antibodies (ANA).smooth muscle antibodies (SMA) or type 1 liver - kidney microsomal antibodies (LKM-1). Titres of 1:40 or more on rodent tissue immunofluorescence are significant in adults.

Treatment of AIH

The 5 year survival of untreated severe AIH is around 50 per cent. and the majority of this mortality occur in the first 6 months of the diagnosis. Combination therapy with prednisolone and azathioprine is the established treatment of severe AIH. Prednisolone is given in a daily dosage of 0.5-

10 mg/ kg/ d and azathioprine in a dose of 1-1.5 mg/kg/d at the initiation of therapy. Response is defined as a fall in serum aminotransferases with histological improvement in the biopsy: however the delay between biochemical and histological remission may vary from 6-18 months.[16]

The histological response occurs in upto 80% patients after 3 years of therapy, with a 5 year survival of 87%. This survival benefit is reduced to 63% in patients who have cirrhosis at the time treatment is started.

OTHER CAUSES OF CHRONIC HEPATITIS

Wilson's disease is an important treatable cause of chronic hepatitis, the evaluation of which requires estimation of serum ceruloplasmin (< 20 mg/dl), a hepatic copper content of more than 250 µg/gm dry weight of liver, and a copper excertion of more than 100 µg/day. In those with a normal serum ceruloplasmin and contraindication to liver biopsy, the oral radiocopper loading test may be performed to exclude Wilson's disease.Treatment can be instituted with D-penicillamine (1gm daily in divided doses), trientine (1-2 gm/d) or oral zinc salts.

Chronic drug hepatitis is characterised by a history of exposure to a hepatotoxic drug, elevated amminotransferase values, or a choloestatic pattern, with high values of serum bilirubin. These abnormalities resolve after withdrawal of the responsible drug. The histology is most commonly a cholestatic lobular hepatitis, but other nonspecific features such as steatohepatitis or granulomatous hepatitis may also be seen. No definitive therapy, other than withdrawal of the incriminated drug, is available.

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Post-MI mechanical complications

P.K. SHAH*; JEFFREY S. GOODMAN**

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ABSTRACT: Three major mechanical complications of acute myocardial infarction-free wall rupture, ventricular septal rupture, and papillary muscle rupture-account for up to 30% of all infarct-related deaths. Survival is unlikely unless the condition is recognized ~~and managed~~ promptly; a high index of suspicion and non-invasive testing are key to early diagnosis. Cardiac rupture can be diagnosed at the patient's bedside with two-dimensional (2-D) echocardiography. Ventricular pseudoaneurysm may be detected with radionuclide ventriculography using the first-pass method or 2-D echocardiography. Septal rupture and papillary muscle rupture can be rapidly confirmed using 2-D echocardiography with Doppler flow studies. Urgent surgical intervention-often the only viable therapeutic option-can improve short term survival in most patients.

Each year in the United States, nearly 1 million persons suffer an acute myocardial infarction (MI). Recent advances in the management of acute. MI, using reperfusion to limit infarct size, have contributed to a significant reduction in postinfarction mortality. This reduction is due, in part, to timely administration of lytic therapy, which may protect against cardiacrupture. In patients who receive reperfusion therapy early after acute MI, the expected mortality at four weeks is less than 5% to 10%.

Pump failure continues to be the major contributor to mortality in acute MI. However, mechanical complications, such as free wall rapture, ventricular septal rupture, and papillary muscle rupture, account for approximately 20% to 30% of all infarct-related deaths.[1-6] Although prompt recognition and intervention can significantly reduce the otherwise high mortality associated with these catastrophic complications, clinical findings may mimic those of other problems. Thus, a high index of suspicion, coupled with the use of noninvasive diagnostic techniques, is key to early diagnosis- and to the patient's survival.

In this article, we will review the clinical aspects of the major mechanical complications of acute MI-ventricular free wall rupture, interventricular septal rupture, and papillary muscle rupture. Our emphasis throughout will be on early diagnosis and management.

FREE WALL RUPTURE

Rupture of the ventricular free wall is one of the most catastrophic complications of acute MI and is associated with a mortality exceeding 90%. While only 1.5% to 8.0% of all infarcts are complicated by free wall rupture, between 8.0% to 24.0% of all infarct-related deaths are attributed to it.

Ventricular free wall rupture is most common in patients with a first MI, in women, in those older than 70 years, and in those with hypertension. The peak incidence of free wall rupture occurs between two and eight days after acute MI, but up to 30% of ruptures occur within the first 24 hours.

Pathophysiology

In patients with ventricular free wall rupture, the infarction is transmural and is secondary to a totally or near totally occluded coronary artey with inadequate collateral circulation . Ventricular rupture occurs with equal frequency in anterior and inferior infarcts; this complication develops most commonly in the left ventricle (LV) and rarely occurs in the right ventricle (RV).

Suggestions that early use of anticoagulation, thrombolysis, ambulation, anti-inflammatory nonsteroidal agents, and corticosteroids contributes to free wall rupture have not been proved convincingly. In fact, there is evidence that prompt coronary artery reperfusion may actually decrease this risk by stopping myocardial necrosis before transmural

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* Director, Professor

** Senior Resident Department of Medicine at Cadars-Sinal Medical Center in Los Angeles. Dr. Shah is director of inpatient cardiology and the coronary care unit. Also Professor of Medicine at the University of California in Los Angeles.

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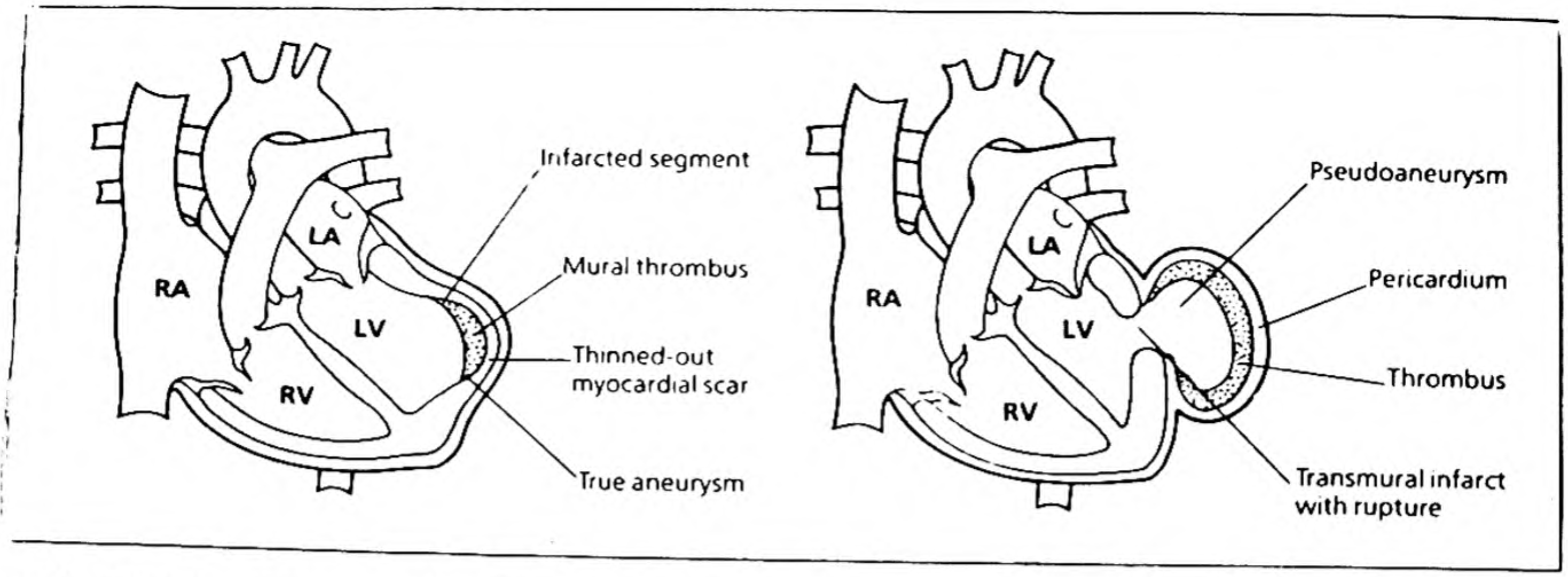

Figure 1- A true aneurysm can be distinguished from a pseudoaneurysm based on a number of findings. A true aneurysm (left) has a wide base, and its walls are composed of myocardium; the risk of free rupture is low. In contrast, a pseudoaneurysm (right) has a narrow base, and its walls are composed of thrombus and pericardium. the associated risk of free rupture is high. Symptoms of a pseudoaneurysm may be similar to those of a true aneurysm. (LA. left atrium; RA. right atrium; left ventricle; RV. right ventricle.) (Adapted from Shah PK, swan HJC: Complications of acute myocardial infarction, in Partmley W, Chatterjee K Philadelphia, JB Lippincott Co, 1987.)

spread, thus sparing a viable epicardial shell that may protect against rupture. It is possible, however, that therapy with anticoagulants or thrombolytic agents could predispose a patient to an increased risk of cardiac rupture if these agents are given after completion of transmural spread of the infarction. This possibility may be an important consideration in the administration of such therapy late in the postinfarction phase.

Diagnosis and therapy

Ventricular free wall rupture may be acute, subacute, or manifested as a pseudoaneurysm.[7-8]

Acute form: The clinical presentation of acute free wall rupture is often quite dramatic, with abrupt onset of chest pain, which the patient may describe as tearing, and which is often indistinguishable from the pain of postinfarction angina or reinfarction. This pain may be preceded by intense agitation, anxiety, & confusion, accompained by marked hypotension; jugular venous distention and vagally mediated sinus or junctional bradycardia. Electromechanical dissociation and death occur rapidly therafter because of hemopericardium and tamponade.[6]

Survival in patients who present with acute free wall rupture is rare and has been reported only in cases in which heroic interventions, including pericardial tap and thoracotomy, were performed at the bedside.

Subacute form: A more subacute form of cardiac rupture has been described in which clinical features of "RV failure," (increasing jugular venous pressure and declining arterial pressure) develop without overt signs of LV failure. These clinical findings result from cardiac tamponade, which develops more slowly than in acute ventricular rupture and may be mistaken for RV infarction.

In patients with this form of cardiac rupture, a high index of suspicion of this complication, accurate diagnosis, and urgent surgical repair can lead to a satisfactory outcome. Suspect cardiac rupture when jugular venous pressure increases in a patient with acute MI who does not have signs of LV failure-Particularly when hypotension and paradoxical pulse are present.

The diagnosis can be established at the patient's bedside with a two-dimensional echocardiogram. The echocardiogram typically reveals pericardial fluid and diastolic inversion of the RV free wall, which indicates tamponade. Occasionally, the actual site of the rupture may be visualized Urgent surgical repair is mandatory, even if the patient's clinical condition improves temporarily with emergent pericardiocentesis, which is generally performed before surgery.

Pseudoaneurysm: This is a rare complication of free wall rupture after acute MI. A ventricular pseudoaneurysm forms from a free wall rupture that is contained by the pericardium. Anatomically, the pseudoaneurysm is not composed of myocardium but of pericardial/epicardial adhesions and pericardium, and it is frequently lined by an enclosed thrombus (Figure 1). Most postinfarction pseudoaneurysms form along the posterior or posterobasal LV wall and communicate with the LV cavity via a narrow neck that is usually half the size of the pseudoaneurysm.

A pseudoaneurysm is often found incidently. However, in some persons, it may present with

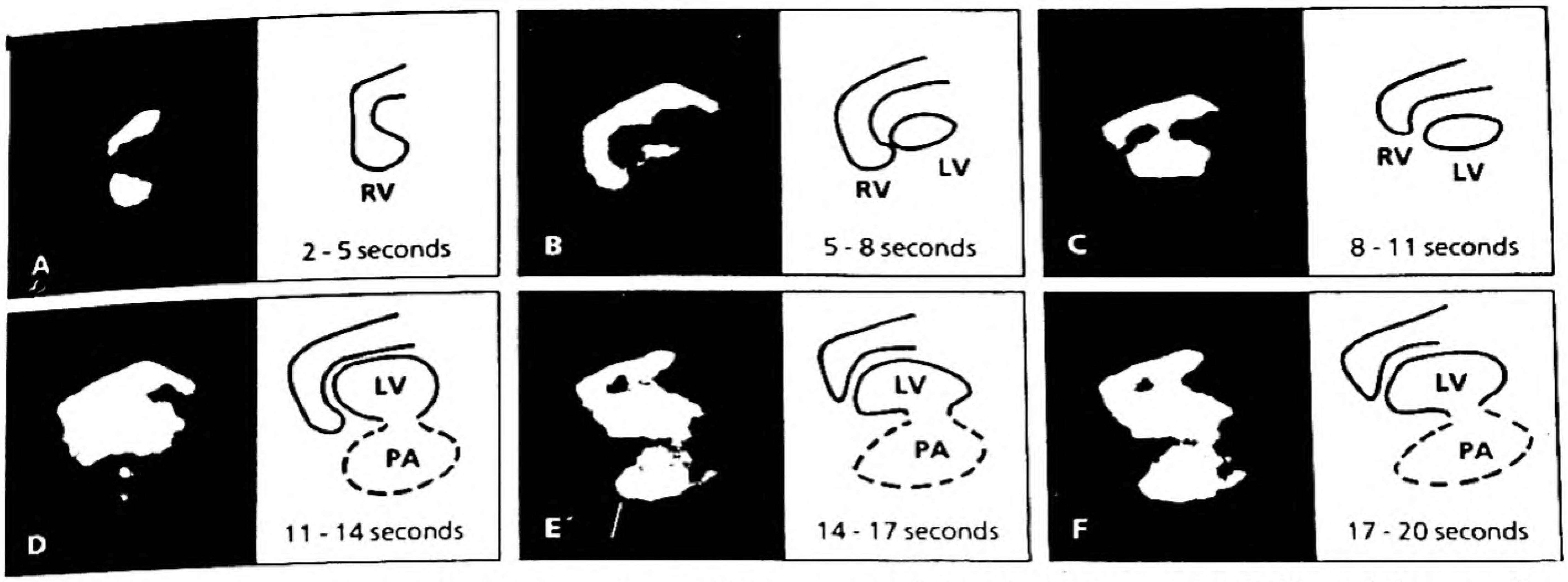

Figure 2 - The first-pass method of radionuclide ventriculography is particularly effective in making the diagnosis of pseudoaneurysm, since sequential filling of the right ventricle (A), left ventricle (B,C), and the pseudoaneurysm (D,E,F) can be visualized. (PA, pseudoaneurysm; RV, right ventricle; LV, left ventricle.)

symptoms similar to those of a true ventricular aneurysm, such as an enlarging cardiac silhouette, congestive heart failure, or ventricular arrhythmias. Systotlic and diastolic murmurs emanating from the flow of blood back and forth across the narrow neck of the pseudoaneurysm have also been described in some patients. Physical examination and chest X-ray findings, and electrocardiographic results may mimic those of a true ventricular aneurysm.

The diagnosis of pseudoaneurysm can be made noninvasively by using a number of imaging techniques, such as two-dimensional echocardiography radionucide ventriculography particularly the first-pass method (Figure 2); Ultrafast computed tomography; and magnetic resonance imaging.

Since pseudoaneurysms are likely to rupture further, resulting in a mortality of nearly 100%, accurate diagnosis is crucial, and prompt surgical repair is almost always warranted, even in the absence of symptoms. Survival after surgical repair is excellent; therefore, it is crucial to consider the possiblility of pseudoaneurysm in postinfarction patients in whom chest x-ray films show an enlarged heart with an abnormal contour, and who have electrocardiographic signs of Q waves and persistent ST segment elevation.

VENTRICULAR SEPTAL RUPTURE

Another devastating complication of acute MI is rupture of the inter-ventricular septum. Like free wall ruptures, most septal ruptures occur between three and five days after acute MI.. However, septal rupture has been reported to develop within the first 24 hours and as late as two to three weeks after MI.

Septal rupture occurs in 0.5% to 2.0% of patients with acute MI. This complication currently accounts for 1% to 5% of all infarct-related deaths.

The natural history of septal rupture is bleak. Without surgical intervention, mortality is 24% at 24 hours after the event, 46% at one week, 80% at four weeks, and 90% or higher at one year.

Pathophysiology

In septal rupture, infarction involves the interventricular septum and usually also includes significant portions of the left and/or right ventricles. This complication occurs with nearly equal frequency in both anterior and inferior infarctions. Septal rupture is most common in multivessel coronary artery disease; however, many instances of septal rupture occur in patients with single-vessel disease.

Rupture of the septum leads to left-to-right interventricular shunt with resultant RV volume overload, increased pulmonary artery flow, and decreased systemic blood flow, which, in turn, results in low systemic cardiac output, hypotension, tissue hypoperfusion, and eventually shock. Depending on the severity of LV necrosis, LV ejection fraction may be depressed, normal, or even elevated as a result of the unloading effects of a left-to-right shunt. RV ejection fraction may be depressed because of acute volume overload, secondary increases in pulmonary artery pressures, and any associated RV ischemic damage, which is commonly seen when septal rupture complicates an inferior wall infarction.

Diagnosis and management

Septal rupture is heralded by the abrupt development of a loud holosystolic precordial murmur with widespread radiation. A palpable thrill can be felt in approximately 50% of patients. In addition, increasing signs and symptoms of congestive heart failure usually develop and are often associated with hypotension and occasionally with frank shock.

The diagnosis of interventricular septal rupture can be rapidly confirmed at the bedside with

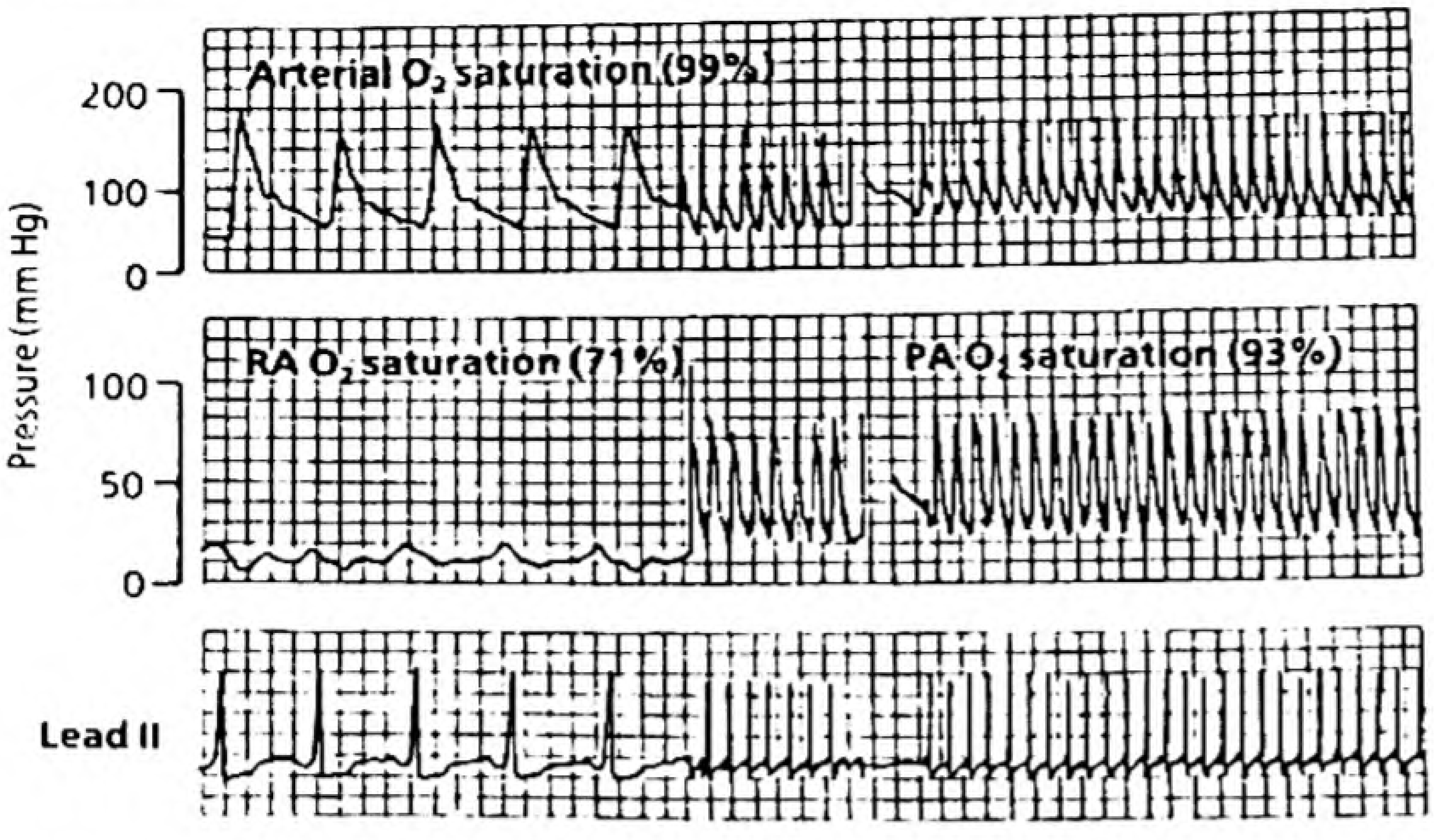

Figure 3 - A definitive sign of post-MI ventricular septal rupture is a step-up of oxygen saturation from the right atrium (RA) to the pulmonary artery (PA) during right heart catheterization. (Adapted from Shah PK, Swan HJC: Complications of acute myocardial infarction, in Parmley W, Chatterjee K (eds): Cardiology. Philadelphia, JB Lippincott Co, 1987.)

two-dimensional echocardiography with Doppler flow studies-especially when color flow imaging is used to demonstrate the septal defect.[10-12] This diagnosis can also be made during right heart catheterization, when a set-up in oxygen saturation of 10% or more from right atrial to right ventricular or pulmonary artery blood is demonstrated (Figure 3). Finally, a diagnosis of septal rupture can be made with first-pass radionuclide ventriculography when echocardiography with Doppler flow studies is not possible.

The definitive management strategy in ventricular septal rupture is urgent surgical repair, which can increase short-term survival up to 50% to 75%. Patients with anterior septal defects appear to have a better surgical outcome than do those with inferior or posterior defects.

Surgery should proceed shortly after the diagnosis is made. (Intervention should no longer be delayed three to six weeks after the acute event.) Before surgery, cardiac catheterization is recommended to assess the severity of coronary artery disease, the presence of any concomitant mitral regurgitation, and the presence of an aneurysm, because these problems may require simultaneous surgical correction.

In addition, temporary clinical and hemodynamic stabilization generally is needed before surgery. This can be achieved by giving vasodilarory/inotropic agents and by the use of intra-aortic balloon counterpulsation.

PAPILLARY MUSCLE RUPTURE

Rupture of the papillary muscle, another catastrophic complication of acute MI-causes acute mitral regurgitation, which is associated with a mortality of nearly 95% at two months in patients who do not undergo surgery.

About 1% of all patients wit acute MI have this complication, which accounts for up to 5% of all postinfarction deaths. Rupture of the papillary muscle usually occurs two to seven days after acute infarction; nevertheless, this complication does develop in up to 20% of affected patients within the first 24 hours after MI.[13,14]

Pathophysiology

Rupture of the posteromedial papillary muscle is much more common than is rupture of its anterolateral counterpart because of a more tenuous blood supply. The mitral valvular apparatus constitutes a complex network that must operate in concert to maintain competence of the normal valve. The mitral valve complex consists of anterior and posterior mitral leaflets, the mitral annulus, anterolateral and posteromedial papillary muscles and their origins on the LV wall, 120 chordae tendineae, and the left atrial wall.

Knowledge of the arterial blood supply and ventricular origination of the papillary muscles is important in understanding the pathophysiology of acute mitral regurgitation. The anterolateral papillary muscle originates at the midportion of the anterolateral left ventricle and has chordal attachments from both mitral leaflets. The posteromedial papillary muscle originates at the union of the middle third of the ventricular septum and the posterior wall. As in the case of the anterolateral papillary muscle, chordae tendineae attachments are received from both mitral leaflets.

The anterolateral papillary muscle receives blood from both the left circumflex artery and the left anterior descending artery or one of its diagnoal branches. The posteromedial papillary muscle must rely on the posterior descending branch of the angiographically dominant coronary artery (right coronary artery in 90% of persons and left circumflex in the remainder) for its blood supply. The nondominant coronary artery occasionally provides sparse additional perfusion.

It is because of this less reliable vascular supply that ischemia, infarction, and rupture of the

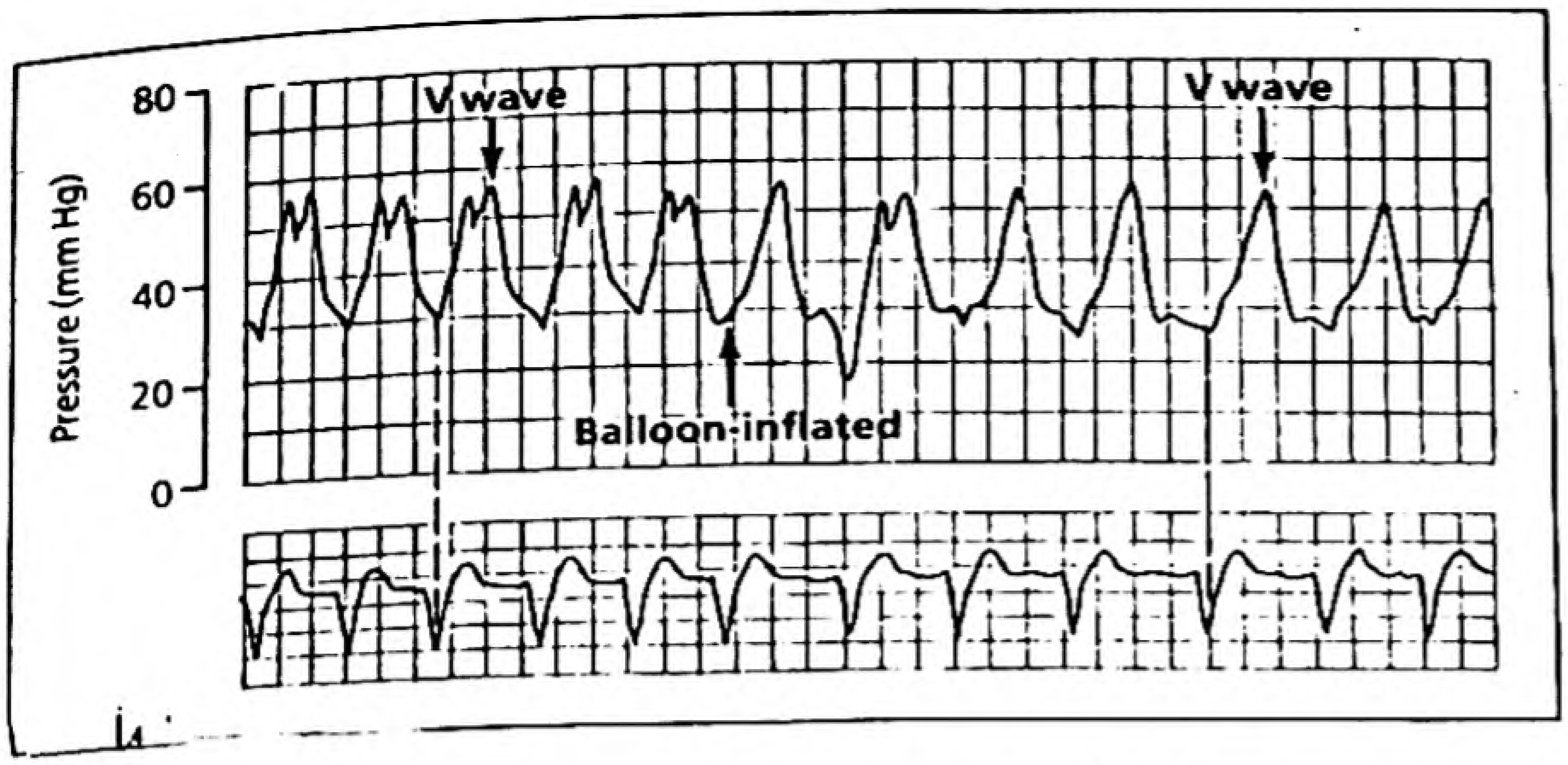

Figure 4 - Large V waves in pulmonary artery and pulmonary capillary wedge pressure tracings are useful in diagnosing severe mitral regurgitation in a patient who has had an acute myocardial infarction. (Adapted from Shah PK, Swan HJC: Complications of acute myocardial infarction, in Parmley W, Chatterjee K (eds): Cardiology. Philadelphia, JB Lippincott Co, 1987.)

posteromedial papillary muscle is five to ten times more common than in the anterolateral muscle. A tenuous blood supply and a high metabolic oxygen demand, similar to that of the subendocardium, predispose the papillary muscle to ischemia and infarction.

Nearly half of all papillary muscle ruptures occur in single-vessel (posterior descending) coronary artery occlusion. Papillary muscle rupture may also complicate either a small or large infarction. Complete rupture of the body of the papillary muscle, which is rare, is usually so catastrophic that survival is unlikely. More frequently, one of the smaller heads of the papillary muscle ruptures.

Diagnosis and therapy

Clinically, papillary muscle rupture causes mitral regurgitation and results in the rapid development of pulmonary congestion or pulmonary edema, hypotension, and shock. A new systolic murmur usually develops, but its characteristics are quite variable, ranging from a loud holosystolic murmur with widespread radiation to the "silent" mitral regurgitation.[15] A papable thrill is rare. (In contrast, a palpable thrill is noted in 50% of patients with ventricular septal rupture.) Concomitant LV and RV dysfunction may occur, depending on the degree of myocardial necrosis.

Chest roentgenography may demonstrate florid bilateral pulmonary edema. On rare occasions, the edema may be distributed asymmetrically to the right upper lobes; this is caused by streaming of the regurgitant jet toward the orifice of the right upper lobe pulmonary veins.

Diagnosis of papillary muscle rupture can be confirmed with two-dimensional echocardiography and Doppler flow studies. Demonstration of a flail mitral leaflet on two-dimensional echocardiography & severe mitral regurgitation on Doppler interrogation is diagnostic of papillary muscle rupture[16].

Right heart catherization is also helpful in making the diagnosis when disproportionately large V waves are noted on the tracings of pulmonary artery and pulmonary capillary wedge pressure (Figure 4). (Occasionally, reflected V waves are seen on the pulmonary artery pressure tracing) However, this finding is not specific for acute mitral regurgitation because it may also be observed in some atients with ventricular septal rupture. Furthermore, mitral regurgitation may coexist with ventricular septal rupture.

Clinical differentiation of acute mitral regurgitation caused by papillary muscle rupture and ventricular septal rutpure can be difficult because of similarities in auscultatory and physical findings. However, additional confirmation can be obtained with two-dimensional echocardiography and Doppler ultrasonography. Clinical signs and diagnostic methods that are useful in differentiation between these complications are listed in the Table.

Management of severe mitral regurgitation ultimately involves left heart catheterization to evaluate valvular and coronary artery patency, followed by surgical replacement of the mitral valve. Vasodilators, inotropic agents, and circulatory assistance with intraaortic ballon counterpulsation may be necessary in the preoperative period.

Circulatory assistance is indicated when the patient continues to be hemodynamically ustable (with low blood pressure and peripheral hypoperfusion) despite administration of inotropic and vasoactive agents.

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Table - Differentiating papillary muscle rupture and ventricular septal rupture

Features/diagnosis	Papillary muscle rupture with acute mitral regurgitation	Ventricular septal rupture
Incidence		
Peak	3 - 5 days after acute MI	3 - 5 days after acute MI
Total	1% of all infarcts	1% to 2% of all infarcts
Relative frequency	More frequent in inferoposterior infarcts (posterior papillary muscle more frequent)	Equally frequent in anterior and inferior infarcts
Signs and symptoms		
Murmur	Usually loud and holosystolic, but may be soft and non-holosystolic or completely absent	Loud and holosystolic with widespread radiation
Palpable precordial thrill	Rare	Present in 50% of patients
Diagnostic techniques		
2-Decho	Flail or prolapsing mitral leaflet	Visualization of defect in septum (with or without contrast echocardiography)
Doppler	Systolic regurgitant jet into LA	Detection of transseptal left-to-right shunt
Radionuclide ventriculography	Normal, increased, or reduced LV ejection fraction with abnormal stroke count ratio	Normal, increased, or reduced LV ejection fraction with abnormal stroke count ratio; left-to-right shunt on first-pass study
PA catheterization	Prominent V waves on PCWP and PA tracing; occasionally, reflected V waves on PA tracing	Oxygen saturation in RV & PA slows step-up compared with RA(PA>RA by > 10% saturation)
LV angiography	Mitral regurgitation visualized	Ventricular septal defect visualized

2-Decho, two-dimensional echocardiography; LA, left atrium; LV, left ventricular; PA; pulmonary artery; PCWP, pulmonary capillary wedge pressure; RV, right ventricule; RA, right atrium.

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Biochemical Assessment of Hepatobiliary Disorders: "Traditional Approaches with New Strategies"

Mohammad Muzaffar Mir; Abdul Rashid Khan; Zaffer Amin Shah*

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In recent years the refinement of imaging techniques such as ultrasonography, percutaneous transhepatic cholangiography, endoscopic retrograde cholangiopancreatography and computerized axial tomography have revolutionised the investigative backup for the more complicated hepatobiliary problems. However such investigations are expensive in equipment and medical time and 75% of cases may be diagnosed by taking a careful history, clinical examination and a few biochemical investigations. New diagnostic means towards chronic liver diseases have been made possible due to recent advances in clinical chemistry. In the light of these advances new strategies for the diagnosis of liver diseases have been devised and recommended by serveral international societies(1,2).

Liver biopsy is the most accurate method to assess the histopathological changes in the liver. However, it is not so simple and easy to carry out the procedure. On the other hand, it is much convenient and useful to carry out a clinical laboratory test which can provide valuable information about the presence and extent of hepatocellular injury. Moreover, much insight into the functional impairment of various metabolic functions of liver and aetiopathogenesis of liver diseases is provided through the conventional liver function tests.

The role of a biochemical set up in the investigation of liver diseases is immense, provided the investigations are carried out with a definite strategy and objective. These objectives are listed below and will be discussed briefly in the following discussion:-

1. To find the aetiology of the disease and its persistence in the liver.
2. To find if the metabolic functions are impaired and compromised.
3. To find out the hepatocellular injury and its severity.
4. To find out the structural status of the liver cells.
5. To find any evidence suggestive of malignancy.

1. To find out aetiology of the disease and its persistance in the liver.

The aetiological factors may be of viral, alcoholic, autoimmune or genetic origins. Hepatic viruses implicated as a cause of chronic hepatitis are of three kinds and these viruses are blood born. Although hepatitis B virus (HBV) has been shown to be a major cause of chronic hepatitis, Non-A, Non-B hepatitis virus is equally important because acute hepatitis caused by it has been shown to progress to chronic hepatitis.

Another causative is the delta virus infection. This may occur simultaneously with HB virus or may be superimposed on a chronic HB carrier state. Delta virus infection may cause asymptomatic HBV carriers to develop chronic active hepatitis (CAH). In case of HBV chronic hepatitis, inflamation of the liver usually flares up prior to seroconversion from HBe antigen positive to anti-HBe positive which subsides afterwards. Signs and symptoms similar to those of acute hepatitis sometimes develop on exacerbation of chronic hepatitis by HBV. HBc antibody of the IgM class is usually low in chronic hepatitis and high in acute hepatitis and thus is a useful marker to differentiate the two types. Patients who show high serum transaminase levels after seroconversion are likely to be suffering from either CAH or liver cirrhosis (LC).

Alcohol is another cause of liver damage. In case of alcoholic hepatitis gamma glutamyl transpetidase (GGT, also known as gamma glutamyl transferase), ratio of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) and IgA are characteristically elevated. Alcoholism being a social taboo in many societies and many patients hesitate to express the same to the clinician. Thus GGT measurement can provide a best marker to monitor and follow up such patients.

The autoimmune origin of chronic hepatitis is characterized by elevated levels of serum globulins and antinuclear antibodies. In many laboratories thymol turbidity test which is nonspecific is still being used for this purpose and could be better replaced by serum electrophoresis.

Ganetic origin: Family history and low levels of ceruloplasmin are diagnostic for Wilson's dis-

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*Department of Clinical Biochemistry and Immunology,
Sheri-Kashmir Institute of Medical Sciences.
Soura, Srinagar-190011 (J&K) India.

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case. Serum iron and iron binding capacity are very useful tests for patients suspected of haemochromatosis. In case of cirrhosis with chronic obstructive lung disease, Alpha-1-antiproteinase deficiency must be considered especially in children. Such genetic disorders can be diagnosed by DNA analysis using techniques like polymerase chain reaction (4). Time is not far away when these investigations shall be routinely put to use.

2. To find if the metabolic functions are imparied and compromised.

The metabolic changes in liver disease result in decrease in the synthetic capacity of liver. This affects the total proteins, albumin, and also the blood clotting factors. The prothrombin time or hepaplastin test is the most sensitive test for hepatic failure. Galactose elimination test, cholesterol, ammonia and ratio of branched chain amino acids (Leu, Ile, Val) to the aromatic amino acid, (tyr, phe) are also useful to study the metabolic derangement in chronic liver diseases (5) serum levels of direct bilirubin, bile acids, GGT, ALP and cholesterol are elevated in cholestasis. For the differentiation of intrahepatic cholestasis from biliary tract obstruction, ultrasonography is the test of choice.

3. The find out the hepatocellular injury and its severity

Serum transaminases are sensitive but not specific tests to establish liver cell injury whereas bilirubin is the most specific but less sensitive. AST and ALT levels do not correlate with the severity of liver cell injury since ALT can be moderately increased even in a healthy man with fatty liver (6). Chronic hepatitis is more accurately diagnosed by liver biopsy than by laboratory tests but serial determination of AST, ALT, total bile acids and gamma globulins are useful to differentiate chronic perisitent hepaitis (CPH) from CAH. CPH is a benign and non progressive disease but CAH may progress to liver cirrhosis which is characterized by altered reconstruction of the lobular parenchyma with prominent fibrosis (7). The derangement of blood flow in liver cirrhosis results in Characteristic laboratory findings like high AST/ALT ratio, high bile acids etc. Some patients with compensated liver cirrhosis may have normal value for the common liver function tests. Many studies have suggested that AST/ALT ratio may be the most sensitive and specific index to differentiate LC from CAH, if the patient is not a drinker. Other test profile will include cholinesterase, ICG (Indocyanine green) retention test, BSP (Bromsulphthalein) excretion test, total bile acids, albumin, gamma globulins and platelet count.

Abnormally high GGT and ALP in a middle aged patient suffering from pruritis and jaundice suggest primary biliary cirrhosis (PBC). The appearance of mitochondrial antibodies with increased IgM level is diagnositc for PBC. GGT, ALP, Cholesteol, Bilirubin and total bile acids are also useful in the follow up of such patients.

4. To find out the structural status of liver cells

For the assesment of structural changes type III procollagen, aminopeptidase, propyl hydroxylase or monoamine oxidase can be used as a marker of fibrotic process in the liver. When CAH progresses to LC and portal systemic shunting develops, the AST/ALT ratio, the ICG retention test, total bile acids, ammonia and gamma globulin increases and platelet count and the ratio of Leu, Ile and Val to Tyr and Phe tend to decrease (8). Hyperammonemia or imbalance of amino acids may lead to hepatic coma. Supplementation of the branched amino acids has been reported to be beneficial to patients with advanced cirrhosis. Simple enzymatic methods for estimation of branched chain amino acids and for Tyrosine have been developed by Taniguti (9) and can be routinely used in clinical chemistry laboratory.

5. To find any evidence suggestive of malignancy

Patients with viral liver cirrhosis frequently develop hepatocellular carcinoma. To follow up such high risk patients ultrasonography of the liver and Alpha-fetoprotein should be studied periodically. Other tumour markers such as ferritin and abnormal prothrombin time are less sensitive and specific than alpha fetoprotein. The Alpha-fetoprotein fequently increase in primary hepatoma. The isozmes of ALP and GGT have been shown to be quite useful in the diagnosis of liver cancer. It has been reported that ALP alpha-1-isozyme has the highest (84.4%) diagnostic accuracy followed by GGT-Alpha-1-isozyme (80.20%), GGT (78.8%) and ALP(69.2%) in the detection of primary liver carcinoma (10). When hepatic metastases are present, the rise in GGT in more pronounced than any other liver enzyme so that if GGT result is normal in a patient with established cancer, liver metastases are less likely to be present.

Address for correspondence:
M. Muzaffer Mir,
Department of Clinical Biochemistry
S.K. Institute of Medical Sciences
Soura, Srinagar-190011 (J&K) India.

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A Survey of the Value of Ultrasound as a Predictor of the Findings at ERCP in a District General Hospital

M.R. Teli*; I. Cobden*

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ABSTRACT: The reliability of ultrasonography (U/S) in suspected biliary disease in a District General Hospital setting is not well documented, nor has the effect of an ERCP service on U/S reporting been audited. Two retrospective surveys were carried out, one beginning in 1986 soon after the appointment of a Gastroenterologist trained in endoscopic retrograde cholangiopancreatography (ERCP) and the other in 1991, of successful ERCP's in which US had been performed shortly beforehand. Results show that US was excellent at predicting malignancy with accuracies of 15/16 (93.8%) and 22/22 (100%) in the two series. US was not very reliable at predicting CBD stones (by reporting either CBD stones, dilatation or both) with a pickup of only 17/28 (60.7%) and 15/31 (48.38%) in the two series. Likewise there was only a moderate success rate in the detection of ERCP-demonstrated dilatation of CBD/biliary tree, of 31/46 (67.4%) in 1986 improving to 41/52 (78.8%) in 1991. The overall performance of U/S in the survey was disappointing and not withstanding improvement seen in the second series, which showed a positive predictive value of 90.2% (46/51), the negative predictive value of only 58.1% (25/43) means that such a report cannot be relied upon to exclude obstructive pathology. There is a need to provide ERCP facilities in most endoscopy units.

INTRODUCTION:

The place of ultrasound (U/S) in the evaluation of pancreatico-biliary disorders is well established. Ultrasonography is a technique which is operator dependent, but sensitivities and specificities of upto 90% and 86% respectively have been reported for the ultrasonic detection of common bile duct (CBD) dilatation and stones (1,2). Most of the published reports come from teaching hospital departments and radiologists who specialise in this field of expertise. The situation may not be comparable in many District General Hospitals. This study was undertaken for two reasons: firstly, to assess the accuracy of ultrasound as a predictor of the findings at ERCP and secondly, to determine whether the introduction of an ERCP service locally, with the possibility of greater feed-back, might lead to improved diagnostic performance for ultrasonography. This was based on a clinical impression that ultrasound reports had improved in diagnostic accuracy over a period of five years since the introduction of ERCP with the appointment of a gastroenterologist trained in the technique.

METHODS:

From 120 consecutive successful endoscopic retrograde cholangio-pancreatography (ERCP) procedures, in which an ultrasound examination had been carried out shortly before and beginning with the introduction of the service in North Tyneside hospitals in 1986, the case records of 85 (70.8%) were available for analysis. A corresponding series of 120 more recent ERCP's (1991-92) produced 94 case records (78.3%) for study. The presenting symptoms, physical findings, results of standard liver function test (LFTs) and the sonographic findings were recorded. The ERCP report was also noted and in addition the radiographs were re-examined to allow for formal measurement of common bile duct diameter at it's widest point. This was done by comparing the measurement with the image of the endoscope (3). A CBD diameter of 10 mm. or less was regarded as normal; above this value, the duct was considered to be dilated (4).

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*Gastroenterology Unit
Preston Hospital North Shields
Tyne & Wear NE29.OLR

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In clinical practice, the principal utility of ultrasound of the biliary tree is in the detection of obstructive pathology requiring drainage, stone removal or other intervention. Therefore, any mention in the ultrasonographic report of dilatation of the intra-hepatic or extra-hepatic biliary system and/or the presence of a stone in the common bile duct was regarded as a "positive" result in this study: conversely, reports were considered "negative" if the biliary system (excluding the gall bladder) was described as normal or was not mentioned specifically (e.g. in some reports commenting only on the liver parenchyma and gall bladder). Thus a true positive result was one where the ERCP demonstrated a CBD calculus, dilatation or both which had been predicted by a positive ultrasound report. A false positive was one in which the biliary tree was normal at ERCP despite a positive ultrasound. Corresponding evaluations were used to define true and false negative results. The sub-groups of patients specifically with CBD calculi or malignant pathology were identified and their results analysed.

The following calculations were made:

1. Accuracy of Ultrasound was calculated as the true positives plus true negatives/all results, expressed as a percentage.
2. Sensitivity was defined as the true positives/true positives plus false negatives, expressed as a percentage.
3. The specificity was defined as the true negative results/true negatives plus false positives, expressed as a percentage.
4. The positive predictive value was defined as the true positives/true positives plus false positives, expressed as a percentage.
5. The negative predictive value was defined as the true negative results/true negatives plus false negatives, expressed as a percentage.

RESULTS:

The findings at ERCP for the two groups of patients are summarised in table 1.

Of the normal ERCP's, ultrasound correctly identified 20/27 (74%) in the first group and 25/30 (83.3%) in the second, i.e. there were 7 and 5 false positive results respectively. The results for patients with a benign abnormality of the biliary tree are summarised in table 2.

For patients with malignancy, ultrasound was highly accurate. In the 1986-1988 patients, one with a cholangiocarcinima was reported as normal. Another report on a patient who turned out to have pancreatic carcinoma was simply that of "oedema of the head of pancreas", but allowing for this to be a true positive report gives an accuracy of 15/16 (93.8%). In the 1991-1992 group, all the malignancies had abnormal sonograms 22/22 (100%).

Taking the overall results for ultrasound as a predictor of ERCP findings, the results are summarised in table 3.

Considering the important sub-group of patients with a gall stone or stones in the CBD at the time of ERCP (i.e. requiring endoscopic sphincterotomy and/or other procedures such as lithotripsy or surgery), ultrasound did not perform well: the results are summarised in figure 1.

Ultrasound performed rather better at detecting dilatation of the biliary tree, as shown in table 4.

DISCUSSION:

Ultrasonography is widely accepted as the initial investigation of choice for patients with suspected biliary tract disease (1,5,6). The ability of ultrasound to assess the presence and nature of obstruction has been discussed in a number of reports (2,7-10). Early publications reported an overall sensitivity of biliary ultrasound of only 50-57% (11,12), but with increasing experience and better equipment the reported results improved in later series (1,2,9,10), with sensitivities reported from 84 to 90%. Most of the published reports have been from teaching hospital departments (1,7-10,13), though in one report U/S was performed at 12 different hospitals with similar results (2).

The North Tyneside hospitals represent an average District General Hospital with a heavy radiological workload for a department which is understaffed according to national guidelines. Sonography is performed by all three radiologists and also by radiographers, with radiologists reporting the results. Bearing this in mind, it is not surprising that the results of sonography are somewhat disappointing. Although there was a good positive predictive value of over 90% in the survey of recent results and a 100% pick-up of malignancy, the negative predictive value of only 50-58% is particularly worrying as it implies a large proportion of "unsafe" negative ultrasound reports.

To be fair, it is likely that the series reported may have given an underestimate of the true success rate of ultrasonography, with two main potential sources of error. Firstly, we have been careful in this study to refer to the sonographic data in terms of their ability to predict the findings at ERCP, not in absolute respect of diagnosis. We accept that ERCP is not necessarily the gold standard. Small calculi can rarely be missed at ERCP (14) and the relevance of duct dilatation without calculi or other pathology is questionable, which is why we have also analysed the results for the sub-group of patients with CBD stones or malignancies. Nevertheless, ERCP is likely to be the most useful procedure to follow ultrasonography in this context and we have shown that the figures in the group with CBD calculi are not very reliable, with a sensitivity of only 55-60% for indicating the need for ERCP (i.e. showing stone or dilatation).

The second concern is that this is a selected group of patients, namely those who have undergone ERCP and does not represent all patients who have had ultrasound scans of the biliary tree. If one considers the patients who underwent ERCP despite a negative sonogram and in whom a CBD stone, for example, was found at ERCP, it is likely that the clinical suspicion was sufficiently strong to override the results of ultrasonography. There are presumably many patients with a lower index of suspicion who have had a negative ultrasound and have not proceeded to ERCP: inclusion of these would probably have greatly raised the true negative rate and affected the percentages. However, we do not know how many cases of CBD calculi may have been overlooked in this group because the features may be a typical, as in some elderly patients (15) or because the responsible clinician had a high threshold for requesting ERCP after negative ultrasound. It is possible to improve the sensitivity of pre-ERCP screening in our patients by incorporating the results of liver function tests. Thus, only four of the twenty-eight patients in the 1986-1988 series with stones and seven of the thirty-one in the 1991-1992 group had normal LFTs and no stones/dilatation in the CBD at ultrasound. If one also includes those patients with a sonographic report of gall bladder stones, then 27/28 and 30/31 patients respectiyely had an ultrasonically-detected abnormal CBD or gall bladder stones or else had abnormal liver function tests. On the other hand, in many cases the liver function abnormalities were minor (e.g. gammaglutamyl transpeptidase 95 IU/L (normal <65) or alanine amino-transferase 54 IU/L (normal <45) and of course gall stones are common, so extending the criteria for ERCP to include all such patients would markedly reduce the specificity of ultrasonography, not to mention greatly increasing the workload of the Gastroenterology Unit with a likely large increase in negative cholangiograms

Our clinical impression of an improvement in ultrasound with time, experience and perhaps feedback from ERCP does seem to be borne out, although the difference is not dramatic and perhaps reflects a dilution effect which might be improved if sonographic skills could be concentrated in one or two specified operators. The logistics of organising this in a small radiology department must be extremely difficult.

In conclusion, this study has shown that the results of ultrasonography in a District General Hospital may not be comparable to those reported from large teaching centres with a concentration of expertise in one or two operators. Detection of malignancy is excellent but CBD calculi will be missed if excessive reliance is placed on negative ultrasonography; clinical suspicion and/or liver function results must be taken into consideration. This has major implications for the endoscopic workload in many districts. Improvements in the staffing of undermanned radiology departments with more focussing of sonographic skills are probably needed to increase the reliability of ultrasound scanning of the biliary tree.

District General Hospitals in Developed Countries are as well equipped if not better than most teaching hospitals in Third World Countries. In Indian sub-continent ERCP facilities are unavailable in most of the hospitals. There is a need of establishing ERCP services in most endoscopy departments to help in diagnosis and management of pancreatico-biliary diseases.

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Correspondence:
Dr. M.R. Teli
Consultant Physician/Gastroenterologist
Louth County Hospital Louth Lincolnshire.
LN 11 OEU (UK)

TABLE 1:

ERCP FINDING	1986-1988	1991-1992
Normal	27 (31.8)	30 (31.9)
Choledocholithiasis	28 (32.9)	31 (33.0)
Other benign	14 (16.5)	11 (11.7)
viz: stricture	2	2
CBD dilatation + no stone	12	9
Malignant	16 (18.8)	22 (23.4)
viz: Ca. pancreas	10	10
Ca. ampulla	2	1
Other - (cholangiocarcinoma/nodal compression - no histology)	4	11
Total	85 (100)	94 (100)

Number (percentage) of patients with ERCP findings in first (1986-1988) and second (1991-1992) study periods.

TABLE 2 - ULTRASOUND PREDICTION OF ERCP FINDINGS FOR BENIGN LESIONS:

ERCP FINDINGS	1986-1988	1991-1992
CBD stone(s) + dilatation	14/22 (63.6%)	15/23 (65.2%)
CBD stone(s) + normal calibre duct	3/6 (50%)	0/8 (0%)
CBD dilatation only	6/12 (50%)	7/9 (77.7%)
Benign CBD stricture	0/2 (0%)	0/2 (0%)

Frequency of true positive ultrasound results for patients in both groups with different benign pathologies.

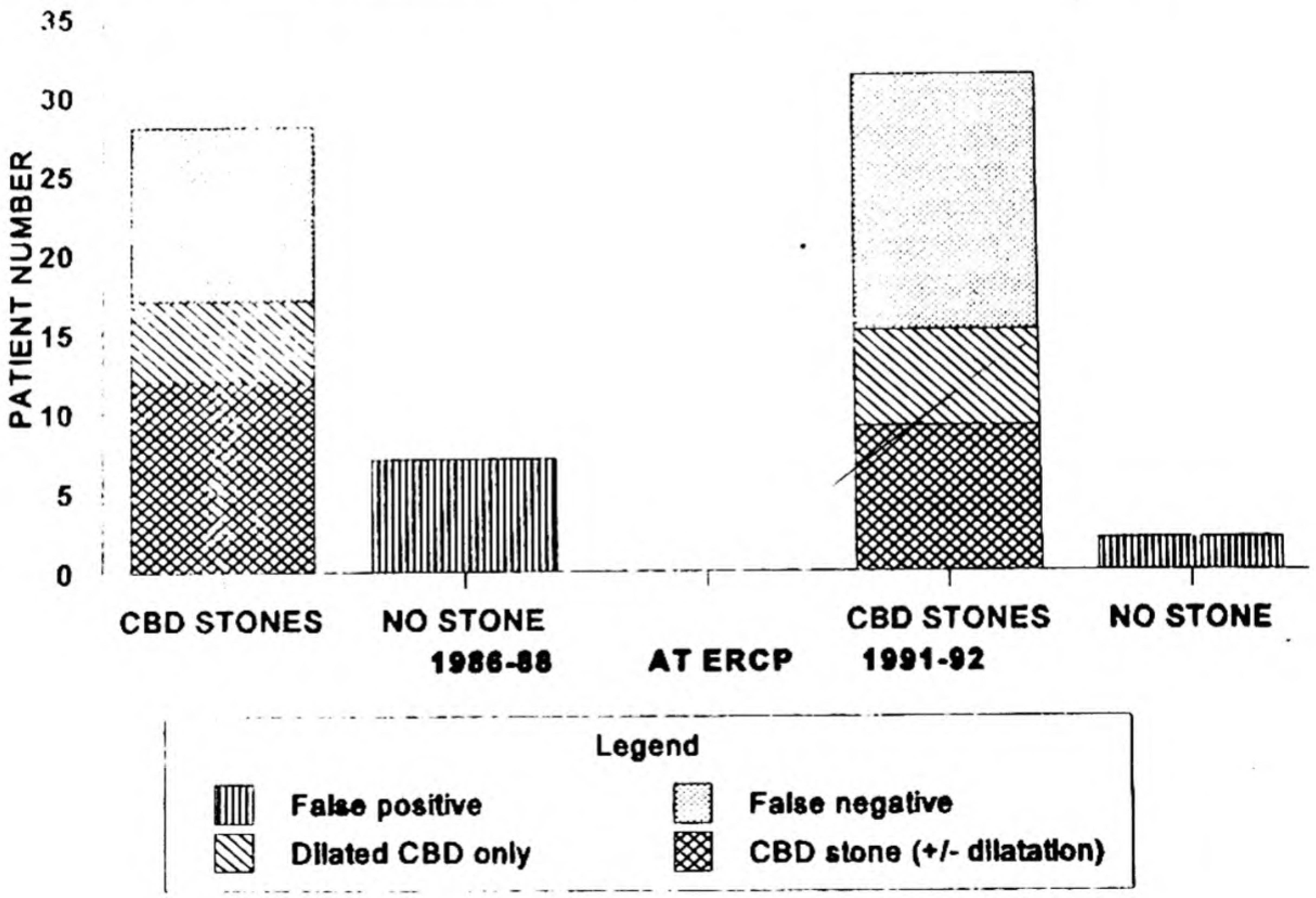
TABLE 3 : OVERALL UTILITY OF ULTRASOUND AS PREDICTOR OF ERCP FINDINGS:

	1986-1988	1991-1992
Accuracy	58/85 (68.2%)	71/94 (75.5%)
Sensitivity	38/58 (65.5%)	46/64 (71.9%)
Specificity	20/27 (74.1%)	25/30 (83.3%)
Positive predictive value	38/45 (84.4%)	46/51 (90.2%)
Negative predictive value	20/40 (50%)	25/43 (58.1%)

TABLE 4 - RESULTS OF ULTRASONOGRAPHY IN PATIENTS WITH A DILATED CBD AT ERCP

	1986-1988	1991-1992
Total patients	85	94
Total dilated CBD (ERCP)	46	52
True positive U/S (dilatation)	31	41
False negative U/S	15	11
False positive U/S	7	6
Accuracy	74.1%	81.9%
Sensitivity	67.39%	78.8%
Specificity	82.05%	85.7%
Positive predictive value	81.57%	87.23%
Negative predictive value	68%	79.24%

Ultrasound Prediction of ERCP for CBD Stones



	1986-88 %	1991-92 %
SENSITIVITY (STONE)	**42.8**	**29.0**
SENSITIVITY (ANY ABNORMALITY)	**60.7**	**48.8**
PREDICTIVE VALUE (POSITIVE)	**70.8**	**93.7**
PREDICTIVE VALUE (NEGATIVE)	**64.5**	**64.4**

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Chemical Composition of Gall Stones

Abdul Majeed Bhat*; Ishtyak Ahmed Mir;
Omar Javad Shad*; Nazir Ahmed Wani*****

Abstract: This study was conducted to know the chemical composition of gall stones and thus assess the possible etiological factors in gall stone formation. Stones from gall bladder and common bile duct of 50 patients were obtained upon surgery and analyzed for different chemical constituents by modified method of Holt and Tronky (1953) [1]. Majority of stones were of mixed cholesterol type followed by pigment stones. Pure cholesterol stones were rare. Residue was found in significant amount in stones and represented the organic matrix of mucopolysacchrides and mucoproteins.

Key words: Gall stones, C.B.D. stones, cholesterol, bilirubin, mucopolysacchrides, mucoproteins.

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Introduction:

Gall stones disease is afflicting a fairly significant portion of human population throughout the world, with a wide variation from region to region. In Pima Indians of North America 70% of women have gall stones by age of 30 years while same percentage of men get it by 60 years of age [2]. On the other hand Masai tribe of East Africa practically have no gall stones[3]. The range of composition difference in gall stones from cholesterol to pigment reflects the different mechanism and etiological factors in its formation, yet the cause of gall stone formation remains obscure. The first step to know this will be to know the composition of gall stones in a particular region and thus the factors causing the precipitation of such components.

We have studied gall bladder and common bile duct stones from 50 patients from Kashmir where the incidence of gall stone disease is about 5% and 12% in general population men and women [4] respectively and gall stone disease accounts for about 21% of total surgical admissions. Gall stone surgery constituted about 18.6% of total major surgical operation in our hospital [5].

Material and methods:

Patients suffering from gall stones disease in the age group of 20 to 80 years were selected from both sexes. They were evaluated fully for presence of cholelithiasis and other disease were ruled out pre-operatively. Stones were collected upon surgery of gall bladder or common bile duct and washed with sterile normal saline. Stones were immediately packed in sterile polythene bags (sterilized by gamma irradiation) and analyzed for chemical constituents by step-wise solvent extraction (modified method of Holt and Tronky [1]).

Large stones were cut sectioned and one cut surface was stained with periodic acid schiff while other surface with alcien blue.

Results:

The majority of patients had stones in only gall bladder (70%), concomitant stones in G.B. and C.B.D was the next common finding (16%), 6 patients had only C.B.D stones (12%) and gall bladders of these patients were removed surgically average 8-20 years back. No patient had CBD stones with a normal gall bladder. One patient (2%) had stones in GB, CBD and hepatic duct.

Cholesterol was the major constituent in majority of stones ranging from 12-98% of dry weight irrespective of their location. Other constituents were proteins, calcium, bilirubin and phosphates (Table-1). The chemical composition of concomitant GB and CBD stones was similar with little difference of constituents measured as percentage of dry weight (Fig. 1 & 2).

Majority of stones in GB & CBD of mixed cholesterol followed by calcium bilirubinate while pure cholesterol were the rare. Mixed cholesterol stones were more in females while calcium bilirubinate stones were more in male population. The highest incidence was found in 5th decade in females followed closely by 4th decade while in males the highest incidence was in 4th decade (Fig. 3).

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*Lecturer.
**Senior Resident.
***Professor and Chairman Surgery Deptt.
S.K. Institute of Medical Sciences.
Srinagar. (Kashmir)

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Stones analysed from common bile duct were of mixed cholesterol in composition. Primary and secondary duct stone were similar in composition, having cholesterol and residue as the major constituents.

On histochemical analysis using PAS and Alcien blue stains the stain positive results were 66.6% and 54.7% respectively in mixed cholesterol stones. In case of pigment stones only 33.3% were PAS positive while none of the stones got strained with Alcien blue (Fig.4).

Discussion:

Gall bladder was the primary site of stones (70%) of patients in our study 16% had both GB and CBD stones. The composition of stones in GB and CBD was identical signifying a similar etiological factor for their formation. Only 2% of our patients had concomitant GB, CBD and hepatic duct stones, all having a similar composition. Nakayama (1968) has also reported GB to be the primary site of stone formation, CBD stones are either slipped small stone from GB or developed denovo in CBD as in patients who under go cholecystectomy [7,8]. It signifies the persistence of the basic etiological factor in those patients who had already undergone cholecystectomy for gall bladder stones [9].

Cholesterol is the major constituent of all types of stones (GB,CBD) ranging from 64% to 70% of dry weight. While calcium and bilirubin were in comparatvely small quantities. Hence majority of stones were of mixed cholesterol in nature. Similar reports have been noted in other studies [10-12] in Western countries. There is a changing state of gall stones disease in Japan, from pigment stones being more common in past to cholesterol stones now-a-days. Factors responsible for such change are not known clearly but may include the rapidly proceeding urbanization and change of the food habits [13].

In pigment stones also, cholesterol was found in significant ratio 19% of dry weight while bilirubin was 13.1% and calcium 15.4% of dry weight. The main bulk of these stones was residue (42.79%) thought to be mucopolysacchrides and other organic matrix.

CBD stones from patients who had already undergone cholecystectomy were of mixed cholesterol type with a little more bilirubin content. The gall stones of these patients were not available for study. CBD stones contained less cholesterol as compared to gall bladder stones from the same patient. This condition favours pigment deposition rather than cholesterol in CBD stones.

Calcium phosphate and proteins were the constituents in very small quantities in mixed cholesterol stones; calcium was found in significant amount in pigment stones signifying a calcium bilirubinate complex. primary and secondary CBD stones were of similar composition, all having cholesterol the major constituent. The finding signifies the persistence of the basic mechanism of stones formation even after removal of gall bladder in these patients [14,16].

Presence of mucopolysacchrides and mucoproteins a constituent was confirmed on histochemical analysis. Source of mucoproteins and mucopolysacchrides is thought to be gall bladder epithelium itself. The mucus substances form an architecture on which the other inorganic constituent get precipitated and solified [14]. Sutor noted an inorganic shell after cholesterol gall stones were chemically dissolved with solutions of chenodexycholic acid and ethylene diaminetetracetic acid (EDTA) [15]. The shell was found to be composed of mucopolysacchrides and mucoproteins.

Table - I

Chemical type of stone and percent composition of dry weight

Chemical Constituent Mg%	Chemical type of stone		
	Pure Cholesterol	Mixed Cholesterol	Calcium Bilirubinate
Cholesterol			
Range	-	28.0-95.0	12.0-27.0
Mean±SD	98%	72.5±16.2	19.8±4.9
Bilirubin			
Range	-	0.2-11.1	7.2-26.1
Mean±SD	0.2	1.8±2.1	13.6±6.1
Calcium			
Range	-	0.1-19.3	4.2-20.1
Mean±SD	1.0	3.8±2.6	15.6±5.4
Phosphate			
Range	-	0.2-5.0	2.1-11.9
Mean±SD	-	2.1±1.0	5.1±2.8
Protein			
Range	-	0.1-6.3	1.2-5.0
Mean±SD	0.4	3.0±1.0	3.2±1.2
Residue			
Range	-	0.1-51.0	31.8-56.0
Mean±SD	0.4	16.6±13.3	42.7±7.8

Fig.1 CHEMICAL COMPOSITION OF BILIARY STONES (ACCORDING TO SITE OF STONE)

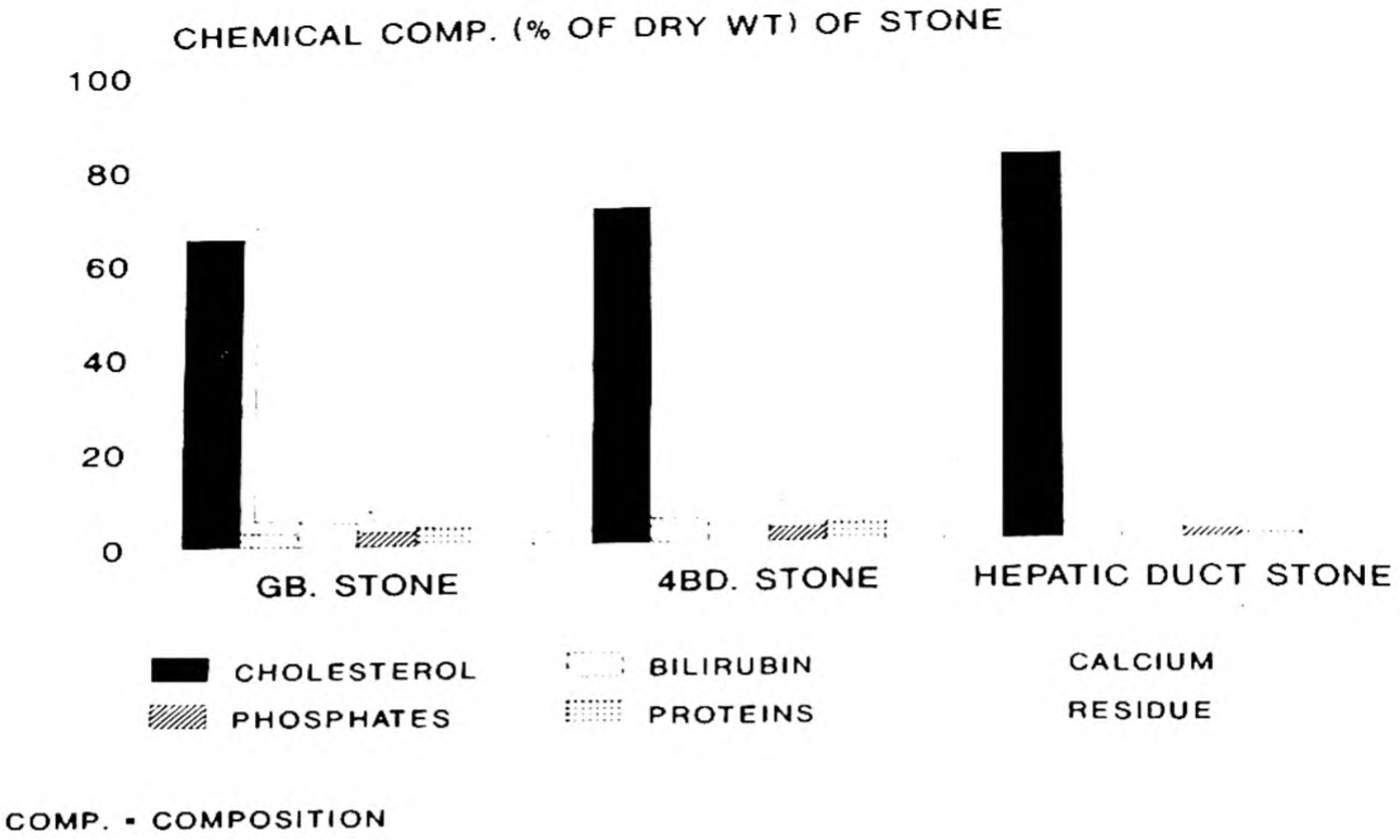


Fig.2 CHEMICAL TYPE OF STONE AND COMPOSITION (% OF DRY WEIGHT)

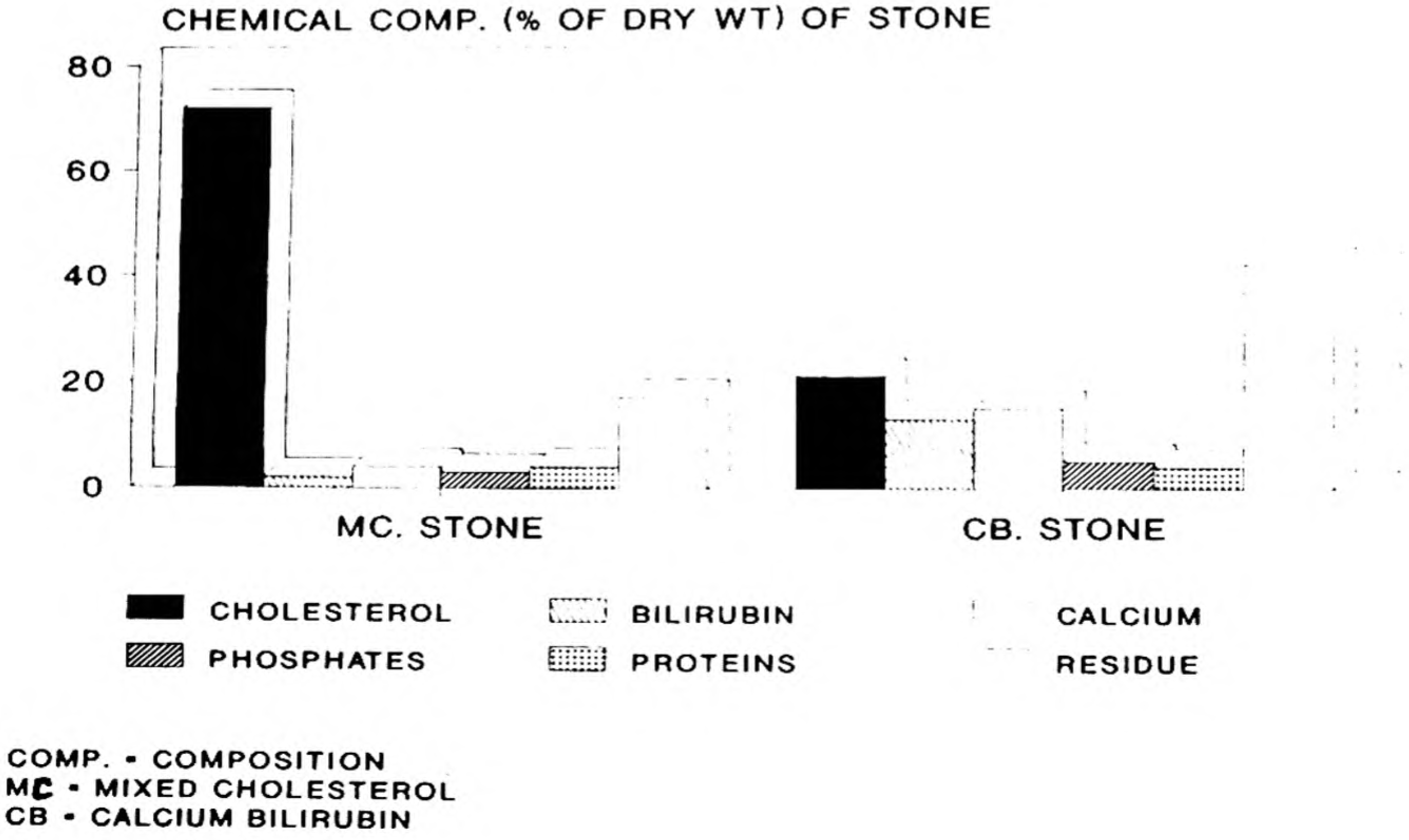
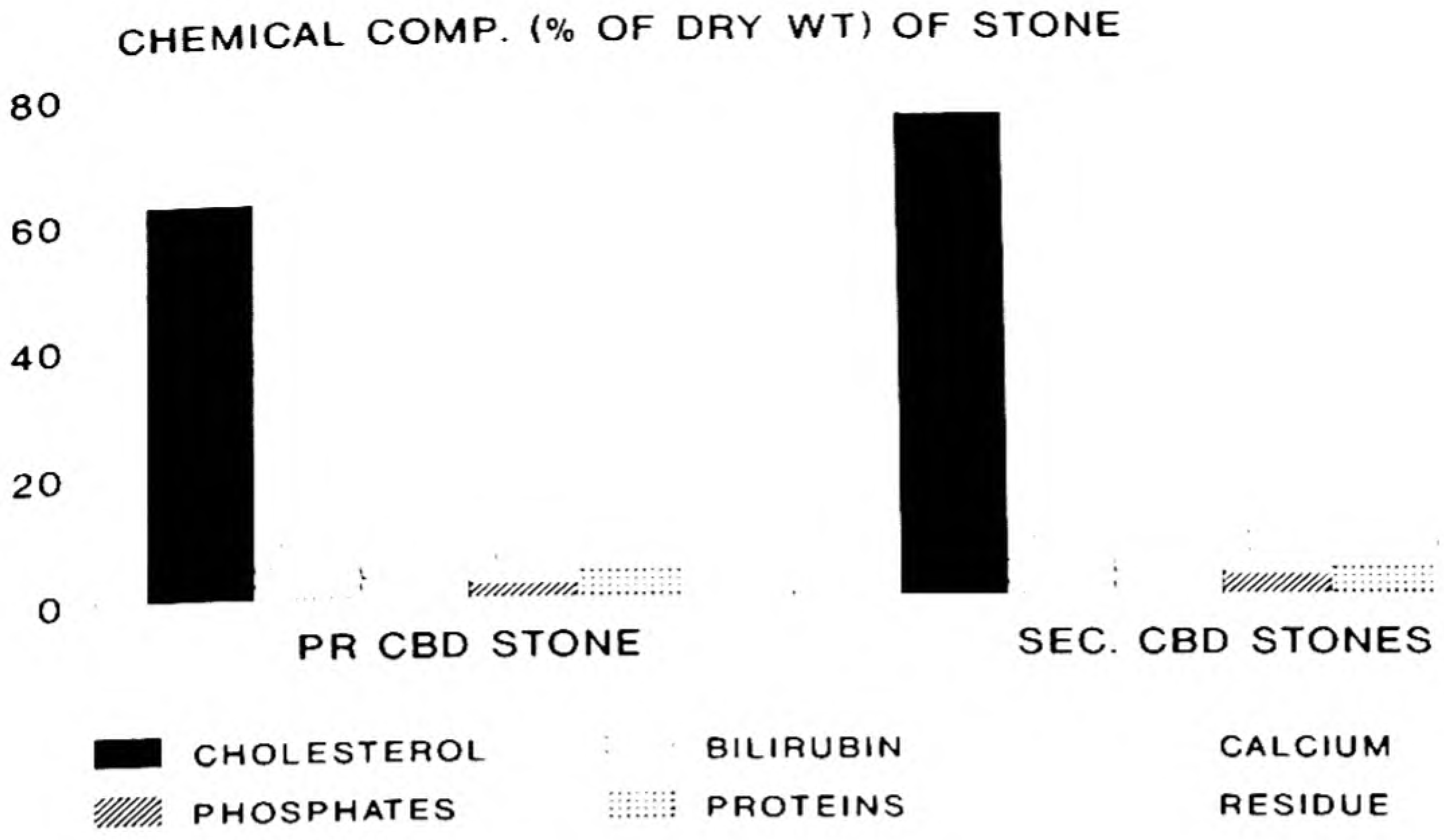
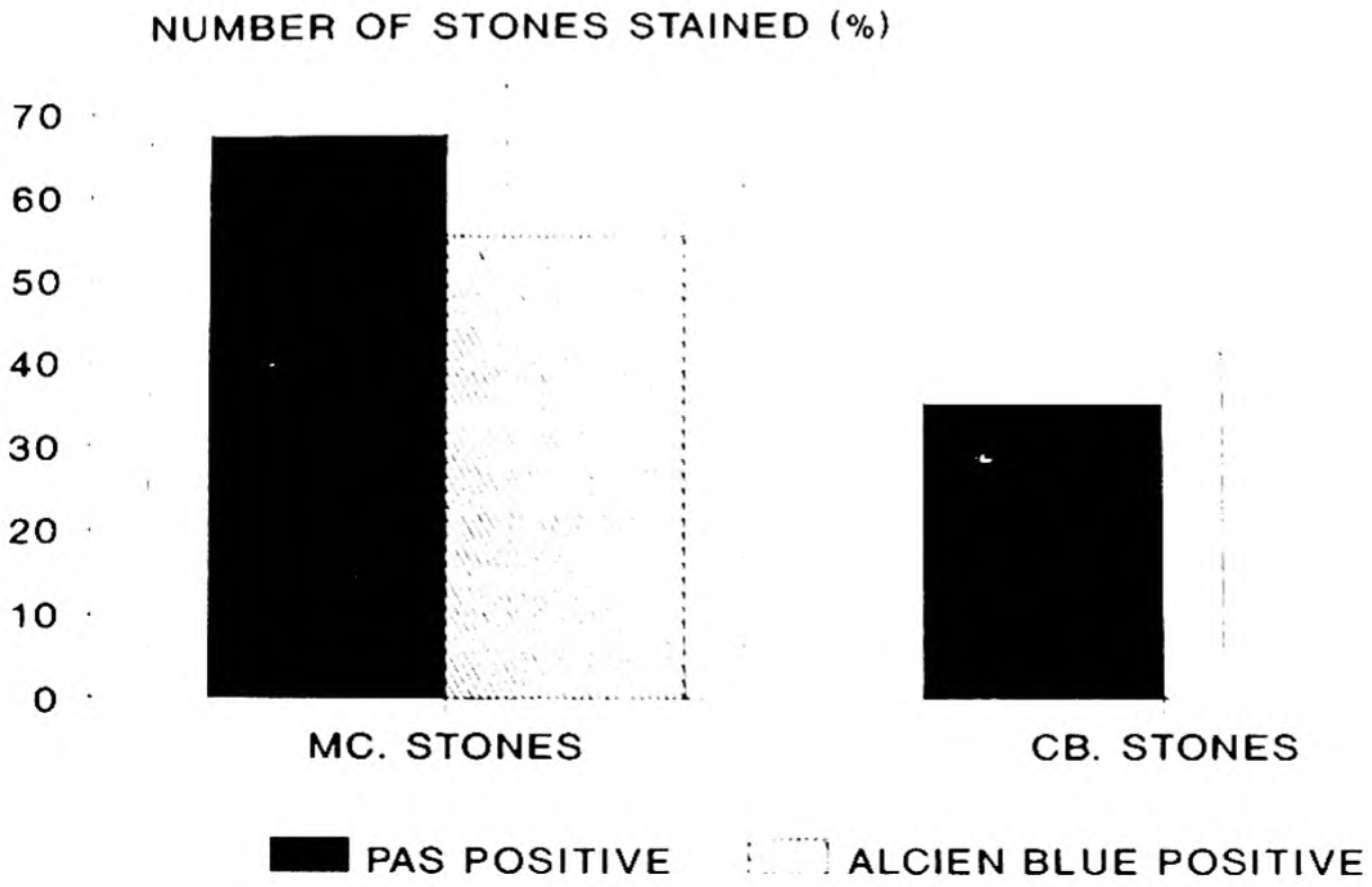


Fig.3 CHEMICAL COMPOSITION OF COMMON BILE DUCT STONES



COMP. - COMPOSITION
PR - PRIMARY
SEC- SECONDARY

Fig.4 HISTOCHEMICAL ANALYSIS OF BILIARY STONES



MC - MIXED CHOLESTEROL
CB - CALCIUM BILIRUBINATE

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Role of Colposcopy in Cervical Cancer Detection

Wazira Khannam*; Saba Hamidi; Asima Rashid*****

JK Practitioner 1996; 3:3 155-156

INTRODUCTION

Hans Hinselman in 1925, made insignificant contribution by inventing an optical device by which the cervix could be examined with sharply focused light and binocular optical magnification, thus pioneering a new field of investigation in gynaecology. "Colposcopy". The great merit of this achievement is not its diagnostic value but clearer understanding of cervical pathology has resulted from its use[2].

Initially Colposcopy and Cytology were considered competitive techniques but now both methods are an essential part of the propramme for screening and early management of cervical neoplasia. Colposcopy is now widely used for evaluation of an abnormal pap smear, for follow up and management of preinvasive lesions.

Colposcopic terminology used to describe the cervical changes includes :

a. Normal colposcopic finding (NCF)
b. Abnormal colposcopic finding (ACF)
c. Unsatisfactory colposcopic finding (UCF)
d. Miscellaneous

MATERIAL & METHODS AND RESULTS

1400 cases were studied colposcopically in the Obstetrics and Gynaecology Department. L.D. Hospital, Srinagar. There were patients who presented with suspicious symptoms like persistent leucorrhea, contact bleeding and postmenopausal bleeding and on clinical examination had and unhealthy cervix. Out of these 1162 (83 percent) had Normal Colposcopic Findings (NCF).

119 patients (8.5 percent) had abnormal colposcopic findings. Out of these 12 patients had invasive carcinoma, 5 patients had carcinoma in situ, and mild to moderate dysplasia was found in 102 patients. Another 119 (8.5 percent) patients had unsatisfactory colposcopic finding where the squamocolumnar function could not be visualized properly or had miscellaneous finding such as inflammatory changes etc.

DISCUSSION

The high incidence and poor prognosis of malignant diseases has prompted increased interest in mass screening programmes for their early detection. Over the last 40 years, the incidence of cervical carcinoma has been markedly reduced due to cytological screening, colposcopy and effective treatment of preinvasive lesions of the cervix. Colposcopy is now established as part of the routine evaluation, to which all women with an abnor-

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*Prinicipal & head Department Obstetrics & Gynaecology

**Post Graduate Student, Department of Obstetrics & Gynaecology Govt. Medical College, Srinagar, Kashmir - 190009 (India)

***Jehlum Valley College of Medical Sciences Srinagar, Kashmir

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mal smear should have access. Colposcopy and directed biopsy of the cervix can provide and accurate localisation and histological diagnosis of premalignant lesions which can be treated by diathermy, cryotherapy or laser ablation or excision.

Lumbery (1958) reported that the rate of carcinoma in situ increased for 4.2% to 27.7% by colposcopy & further he found that colposcopy was negative in 3% [1]

Javeheri & Fijgin (1980) in their study of 930 patients found that 898 patients had satisfactory colposcopic findings & 32 patients had unsatisfactory colposcopy. Colposcopy was accurate when compared to the histology of the colposcopically directed biopsy in 91%[2]

Upreti et al (1981) studied 1000 patients attending gynaecological and antenatal OPD by cervical smears. 200 patients showed abnormal smears, these were then studied by colposcopy. Out of these 200 colposcopies, 45% had abnormal transformation Zone, 6% atrophic, 21% erosion, 8% inflammatory, 13% normal colposcopic findings and 7% unsatisfactory colposcopic findings.[3]

Study of 100 patients with cervical erosion was done by Bandi et al (1985) using colposcopy. In these 6 cases of carcinoma were detected all in age group of 40 and above. All of them showed atypical transformation zone on colposcopy.[4]

With colposcopy, cervical lesions are visualised by proper illumination and magnification of the cervix. The squamocolumnar junction can be visualised, which is considered the "Key point" for the origin of virtually all cervical squamous dysplastic in situ and invasive lesions. With early detection and proper management of precursor lesions, progression to invasive carcinoma can be prevented to a great extent. It has been estimated that the risk of progression to invasive carcinoma with (a) CIN I is approximately 1% (b) CIN II is approximately 5% and (c) CIN III, greater than 12%.

The use of colposcopy in addition to cytology for screening reduces the false negative rate for cytology alone. Together the two techniques have a higher degree of accuracy. Giles et al (1988) screened 200 asymtomatic women both cytologically & colposcopically for evidence of CIN. The prevalence detected by cytology alone was 5% but that detected by cytology & colposcopy together was 11%.[5]

Colposcopy accurately localizes the abnormal areas of cervix from which a directed biopsy can be taken and correct diagnosis can be made. Aggarwal (1989) studied the role of cytology and colposcopically directed biopsies in various lesions of the cervix. Out of the 240 cases studied, cytology with colposcope showed an accuracy of 73% but cytology with colposcopically directed biopsy showed an improved correlation of 89.7 percent.

Human papilloma virus (HPV) of the female genital tract is now recognised as a major risk factor for development of neoplasia. With the combined investigative modalities of cytology, colposcopy, histopathology it is clear that precancerous changes and HPV are closely associated Carmichael & Maskins(1989) studied 235 women with mild to moderate cervical dysplasia associated with a HPV infection, at 6 monthly interval at the colposcopy clinic. 130 patients had conversion to normal and 9 patients (5.5%) had an increasing cervical dysplasia.[7]

The use of colposcpy, in addition to cervical smears, as a screening technique for cervical cancer seems attractive as it identifies more women with cervical intraepithelial neoplasia but is not feasible as it would place considerable demands on resources and it would not be possible to adequately examine all women, hence cytology is now accepted as an effective screening method and colposcopy as the appropriate diagnostic technique for the evaluation of an abnormal pap smear.

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Survival in Water Intoxication
- A Case Report

S. Banzal*, F.A. Bhat*, H.S. Rissam, Jamal A.*****

ABSTRACT: We report a case of young menstruant, psychiatric patient, who after excessive water intake, developed severe symptomatic hyponatraemia. She developed convulsions, decerebrate rigidity and coma. She made full recovery after intensive management. The case is being reported as survival in such cases is not common.

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INTRODUCTION

Hyponatraemia is a frequent electrolyte abnormality seen in hospitalised patients. It is usually dilutional, although total body water and extracellular volume may be high, low or normal.[1]

Patients with hyponatraemia may remain asymptomatic or may present with central nervous system manifestations ranging from headache, sensory impairment, inappropriate behaviour, hallucinations, sphincteric incontinence, decerebrate posture, coma and death if not treated aggressively. Outcome is worst in menstruant women and children.[2]

We present a case of a young menstruant psychiatric patient who developed severe hyponatraemia due to excessive water intake, which led to serious CNS complications. She was given intensive management leading to complete recovery.

CASE REPORT:

M.O. 30 years old menstruant woman was a known case of schizophrenia on antipsychotic drug therapy. She was referred to our Casualty with the diagnosis of subdural haematoma. There was history of repeated generalised tonic-clonic convulsions, during which she had fallen down and sustained head injury. Following that, she had three vomitings and loss of consciousness. Few hours before the onset of these problems. She had drunk about five litres of water during psychotic episode. On arrival, patient was deeply comatosed with laboured breathing, R/R 24/ minute, pulse 68/ minute, BP 130/80mm of Hg. There was an injury mark on her forehead. She had decerebrate posture with semidilated and sluggishly reacting pupils. Plantars were bilaterally extensor. There was no other focal neurological localising sign. Neck was soft with negative Kernig,s sign. Other systemic examination was unremarkable.

Immediate endotracheal intubation and Ambu bag ventilation were done. Blood was collected for urgent biochemical examination. She was suspected of having sustained subdural haemtoma due to head injury after her fall. Because of this suspicion she was given 200ml of 20% i/v mannitol and 10gm dexamethasone. CT Brain was done, which showed no evidence of haemorrhage, infraction or any other abnormality.

Laboratory investgations showed serum sodium 105 mmol/l, serum potassium was 3.6 mmol/l, BUN 1.1 mmol/l, S. Creatinine 33 umol/l, S. Cholesterol, S.triglyceride, random blood glucose, S. Cortisol and Serum T3, T4 values were normal.

MANAGEMENT:

Patient was mechanically ventilated in the Intensive Care Unit, with comprehensive biochemical, haemodynamic, respiratory and neurological monitoring (Table). Basal central venous pressure was 13cm of water. Hyponatraemia was reconfirmed by 2nd sample. As patient's serum sodium was 105 and correction was targeted at 130 mmol/l, there was a deficit of 25 mmol/l. It was planned to fill this deficit at a rate of 1 mmol/hr i.e. in 25 hours. She was started on hypertonic saline (3%), at a rate of 80ml per hour through infusion pump. She was also given stat 40mg i/v Frusemide. Within next 3 hours, she passed 3.3 litres of urine & her serum sodium leve rose to 119 mmol/l. At this stage because of this very rapid correction(i.e 14 mmol in 3

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*Medical Specialist,
**Senior Consultant Cardiologist
***Chief of Internal Medicine
Department of Medicine
King Fahd Central Hospital
PO Box 204, Gizan
KINGDOM OF SAUDI ARABIA.

Received & accepted

hours). hypertonic saline was discontinued and patient was maintained on 0.9% normal saline at the rate of 20ml/hr. By 5 hours urine output was 5.3 litres. CVP decreased to 9cm and serum sodium rose to 125meq/l. By 6PM (19hours), total urine output was 6.4 litres. CVP 8cm and S. Sodium 132 mmol/l. By next day 6 AM (31hours) she had passed total of 7.8 litres of urine. CVP came down to 6cm and serum sodium rose to 136 mmol/l.

Meanwhile BUN and S. creatinine which were below normal range, because of dilution, showed concomitant rise towards normal range (Table). Patient's sensorium improved gradually and she was weaned off the respirator by 5th day. On repeat CT Scan brain was normal. On the 6th day she was fully conscious, with better parameters and was extubated. Later on, she was put under the care of psychiatrist for managing her underlying psyschiatric disorder. By 2 weeks she was on her own and discharged in almost fit shape.

DISCUSSUON:

In 1920, Weir & Co-Workers have described water intoxication in humans, one of their patients drank 5.25 litres of water after administration of pituitary extract. He excreted 800ml of urine, became very ill, nauseated and developed headache.[3]

Hyponatraemia may be chronic and asymptomatic or may be acute and symptomatic. Kidneys have large capacity to excrete free water (upto 17-18 litres/day) so primary polydipsia alone is rarely a cause of symptomatic hyponatraemia.[4] But, polydipsia can cause symptomatic hyponatraemia in individuals with psychiatric illness.[5] 3-5% of institutionalised patients with mental illness develop hyponatremia due to episodic polydipsia.[6] These psychiatric patients have some abnormality in water excretion such as syndrome of inappropriate anti-diuretic hormone (SIADH).[7,8] Antipsychotic drugs, as such, cause hyponatraemia, which may be exacerbated by episodic polydipsia.[9,10] SIADH, firstly described by Schwatz et al in 1957 in a patient with bronchogenic carcinoma,[11] is the most common cause of hyponatraemia in hospitalised patients, particularly in post-operative cases.[12] Young, healthy menstruant women are at particular risk of developing symptomatic hyponatraemia and respiratory arrest, neurological sequalae and death.[1,13,14] Relative risk of death or permanent brain damage is reported to be 28 times greater in women that in men and this risk is as high as 26 times among menstruant women than among post-menopausal.[2] Impairment of brain cell volume regulatory mechanism and sex hormones (estrogens) are thought to play a role in the females for poor adaptation of their brain cells to hyponatraemia.[13]

Patients with AIDS, hypothyroidism and Addison's disease are also prone to get hyponatraemia for various reasons. Starvation may predispose subjects with primary polydipsia to the development of hyponatraemia.[15]

When serum sodium rapidly goes down to 120 mmol/l, patients may present with headache, nausea and vomiting as early manifestations. When not treated in time, they may develop abnormal behaviour,somnolence, seizures, decorticate and decerebrate postures and coma. Mortality rate is as high as 50% in advanced cases.[1,2] The autopsy findings show cerebral oedema which can be detected by CT Scan and MRI.[14,16]

The diagnosis of hyponatraemia is mostly made from routine laboratory tests. Severe hyponatraemia needs immediate therapy. Isotonic saline infusion is indicated if the patient is volume and sodium chloride depleted. In SIADH, associated with volume expansion, treatment with hypertonic saline alone may not be effective, because the administered salt is excreted rapidly in relatively concentrated urine. In these patients, normal saline or hypertonic sa-

Date	Time	Hrs. since admission	Total urine output (cc)	CVP (cm)	Na (mmol/l	S.K. (mmol/l)	BUN (mmol/l)	S.Cr. (Umol/l)
11.3.14	11pm	0	0	13	105	3.6	1.1	33
12.3.14	2am	3	3,300	9.5	119	3.3	1.1	36
	4am	5	5,300	9.0	125	4.0	1.2	37
	6am	7	5,700	9.0	126	4.1	1.4	43
	6pm	19	6,400	8.0	132	4.9	1.9	41
13.3.14	6am	31	7,800	6.0	136	3.9	3.4	49

Table showing the CVP and blood biochemistry parameters and urinary output noted against the time in hours since admission.

line must be combined with frusemide.[17]

This patient, a young menstruant female, relatively starved, schizophrenic, on antipsychotic drugs, had excessive water intake, thus had an ideal setting for developing severe symptomatic hyponatraemia with high risk of morbidity and mortality. Her convulsions, subsequent fall and head injury compounded the clinical picture, making the possibility of subdural haematoma, the referral diagnosis. Quick respiratory assistance, immediate CT Scan to exclude subdural haematoma and early diagnosis of hyponatraemia as the underlying cause of her convulsion and coma led to appropriate correction therapy. Initially, correction of 1 mmol/hr was planned but the serum sodium level came up within 5 hours to 125 mmol/l; possibly contributed by rapid diuresis, as she passed 5.3 litres urine in this much time. As against reported risk of Central Pontine Myelenolysis by rapid correction of hyponatraemia; no such complication developed in her.

Presently, research workers feel that the rate of correction is not a factor in the genesis of hyponatraemic brain damage. More than 160 patients with rapid correction of symptomatic hyponatraemia have been reported without any morbidity, which suggests that rapid correction of hyponatraemia is a safe and effective measure to circumvent morbidity and mortality.[1,13]

Our patient, inspite of all above odds against her, made a full recovery.

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- A Case Report

N.A. Bhat* (FRCS)

or a combination of two and chemotherapy.

The traditional treatment for carcinoma of external auditory meatus involving middle ear is radical mastoidectomy and radiotherapy producing a 5 year survival rate of about 25% overall (Convey: 1965, Lewis: 1975). Radiotherapy used as the only treatment can produce a 5 year survival rate upto 29% (Goodwin & Jesse, 1980). In gross terms these results are not exceeded by those of a 5 year survival rate from temporal bone resection of 27% (Lewis, 1975) in those cases with extensive disease.

We choose to treat our patient with primary radiotherapy because of her age and general condition. Our main aim was palliation against pain which we seem to have achieved as the patient is almost pain free now after 3 months of her completion of radiotherapy.

Moertel et al (1961) found an increased frequency of multiple primary malignant neoplasms, indicating that five percent of patients who have had a malignant neoplasm will develop other primary neoplasms. Our patient had carcinoma of right breast treated with surgery 12 years ago.

CONCLUSION

Carcinoma of the ear is very rare but high clinical suspicion will help in early diagnosis and treatment. This will help in improving the overall survival rate which still continues not to be good.

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Kartagener's Syndrome with Simple Hepatic Cyst

S.A. Kadla*; S.A. Khan**; M. Agaz***; M.A. Wani***;
T. Hussain****; S. Rashid****; J. Basu*; G.M. Malik****

Abstract: A 22 year old female was admitted with history of recurrent upper respiratory tract infections since her childhood. In addition there was history of progressive nasal discharge and dysphnoea of nine months duration. Radiological imaging revealed destrocardia, bronchiectiasis, situs inverses, sinusitis and a cyst in morphological left lobe of liver. Anterior rhinoscopy revealed nasal polyp. This case represents an unusual case of Kartagenar's syndrome associated with simple hepatic cyst.

Key Works: *Kartagner's syndrome, Dextrocardis, Sinusitis, Situs inversus, Nasal Polyp, Cilia*

Abbreviation: *Kartanger's syndrome, Mucociliary transport*

JK Practitioner 1996; 3:3 162-164

Introduction :

Kartagener's syndrome (KS) has been described as the triad of bronchiectiasis, sinusitis and situs inversus[1]. Patient with this syndrome lack mucociliary transport[2,4] and have immotile cilia[5] and immotile sperms[3,5,6]. Electron microscopy of cilia[2,3,6] and sperm[5] from these patients showed that the dynein arms are missing. Their absence has been reported to be the underlying abnormality responsible for this triad of disease[3,5]. It has been suggested that the existence of such an ultrastructural abnormality be renamed the "immotile cilia syndrome"[3]. The dynein arms contain almost all of the ATpase activity necessary for motility of cilia[7]. Ciliary immotility causes abnormality in mucociliary transport (MCT) and would account for diffuse bronchiectiasis and sinusitis. Dextrocardia and situs inversus can be explained by failure of normal rotation of primitive organs during embryogenesis due to defective cilia[5].

No case of K.S. with hepatic cyst has been reported till now. The present paper concerns one such patient who had K.S. with simple intrahepatic cyst in one of the lobes of liver.

Case report:

A 22 year old female born of non-consanguinous marriage, was admitted with one year's history of cough associated with expectoration of mucoid to mucopurulent sputum amounting to 50-100ml per 24 hours. The expectorate occasionally would smell putrid, but was never associated with blood. Associated with cough was history of progressive dyspnoea recurrent headaches, and nasal blockade of nine month's duration. There was history of recurrent cough and rhinitis like illness since childhood. There was no past history suggestive of whooping cough, tuberculosis or otitis media. She was vaccinated only against small-pox during her childhood. There was no family history of twin births. clinical examination revealed an average built young right handed female with normal general physical examination. Her chest examination revealed percussion note to be dull on right side from 3rd to 5th intercostal space anteriorly along midclavicular line while below right 5th intercostal space, the percussion note was tympanitic. Percussion note was dull below left 5th intercostal space anteriorly along mid-clavicular line. Cardio-vascular- Examination revealed features of dextrocardia, that is, heart sounds were best heard on right side of chest at the areas corresponding to normal auscultatory areas on left side of chest. There were no murmurs or pericardial rub. Her abdominal examination was normal except right hypocondrium revealed tympanitic percussion note. Her CNS examination was normal.

Anterior rhinoscopy revealed nasal polyps on left side. Posterior rhinoscopy was normal. Her throat and ear examination were normal.

The haemolobin was 13.9gm/dl. TLC 7200. with 70% polymorphs. 24% lymphocytes. 2% esonophils, 2% monocystes the platelet 3 lakh per mm^3 with a normal peripheral blood smear. The serum urea was 32mg/dl. the creatinine 0.9mg/dl. the sodium 138mmol/l. the potassium 4.2mmol/l. liver function tests & the liver enzymes were normal. The sputum smears were negative for acid fast bacilli.

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* Postgraduate student, ** House physician
*** Registrar, **** Lecturer
*****Associate Professor & Head G.I.T. Division
Department of Medicine
Medical College Srinagar.
Kashmir 190 010 India.

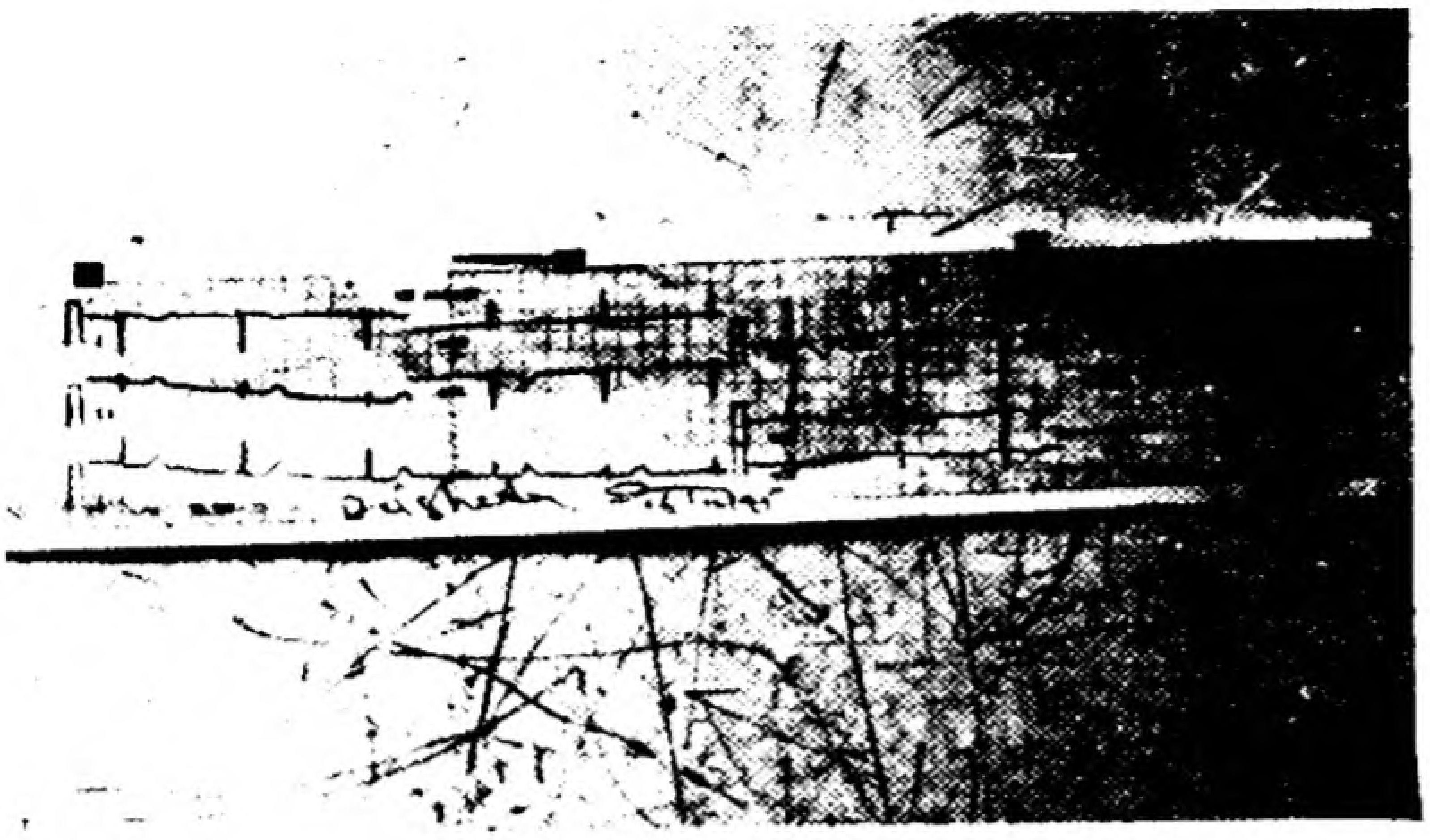
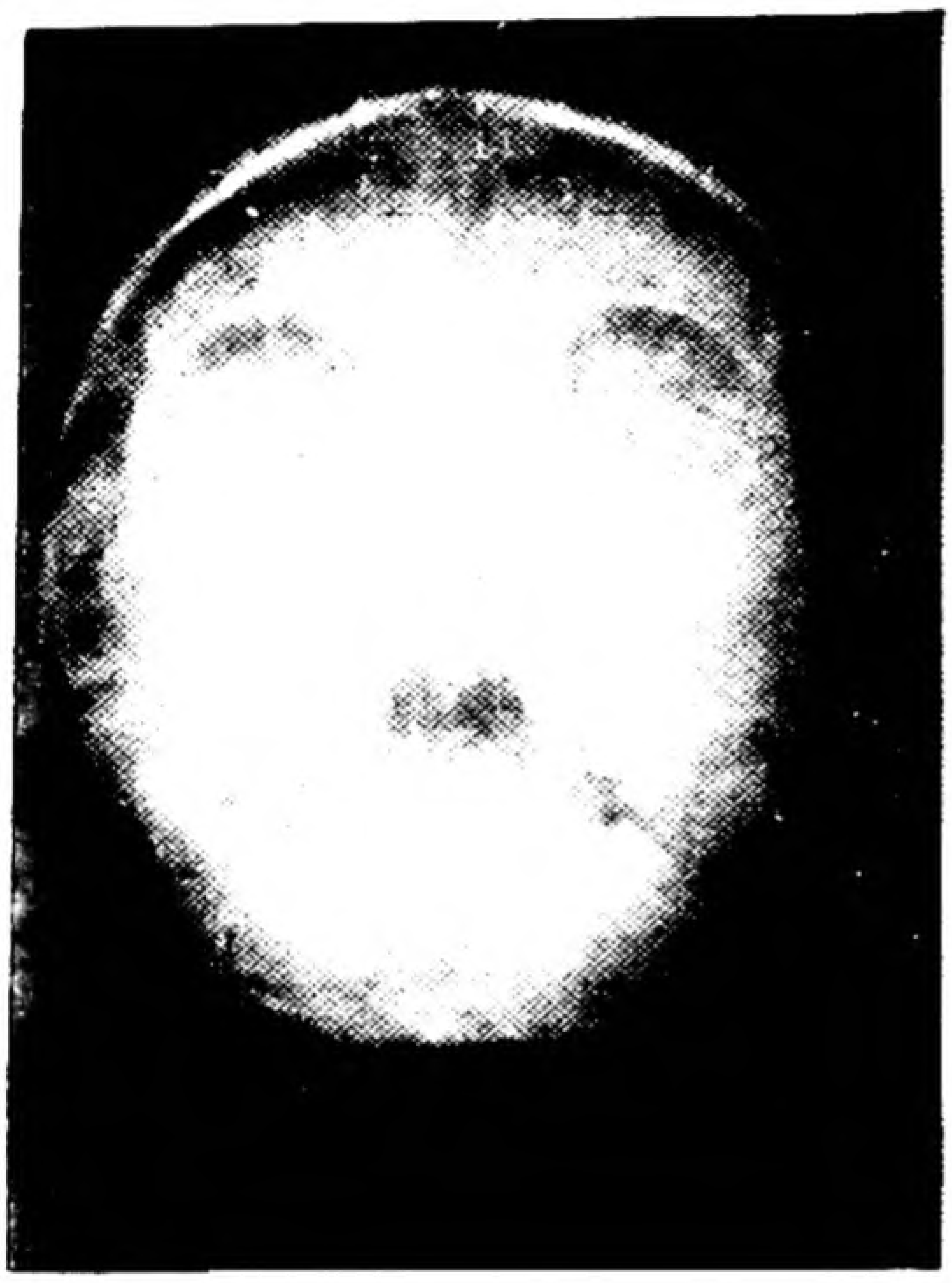


Fig. I

Fig. II



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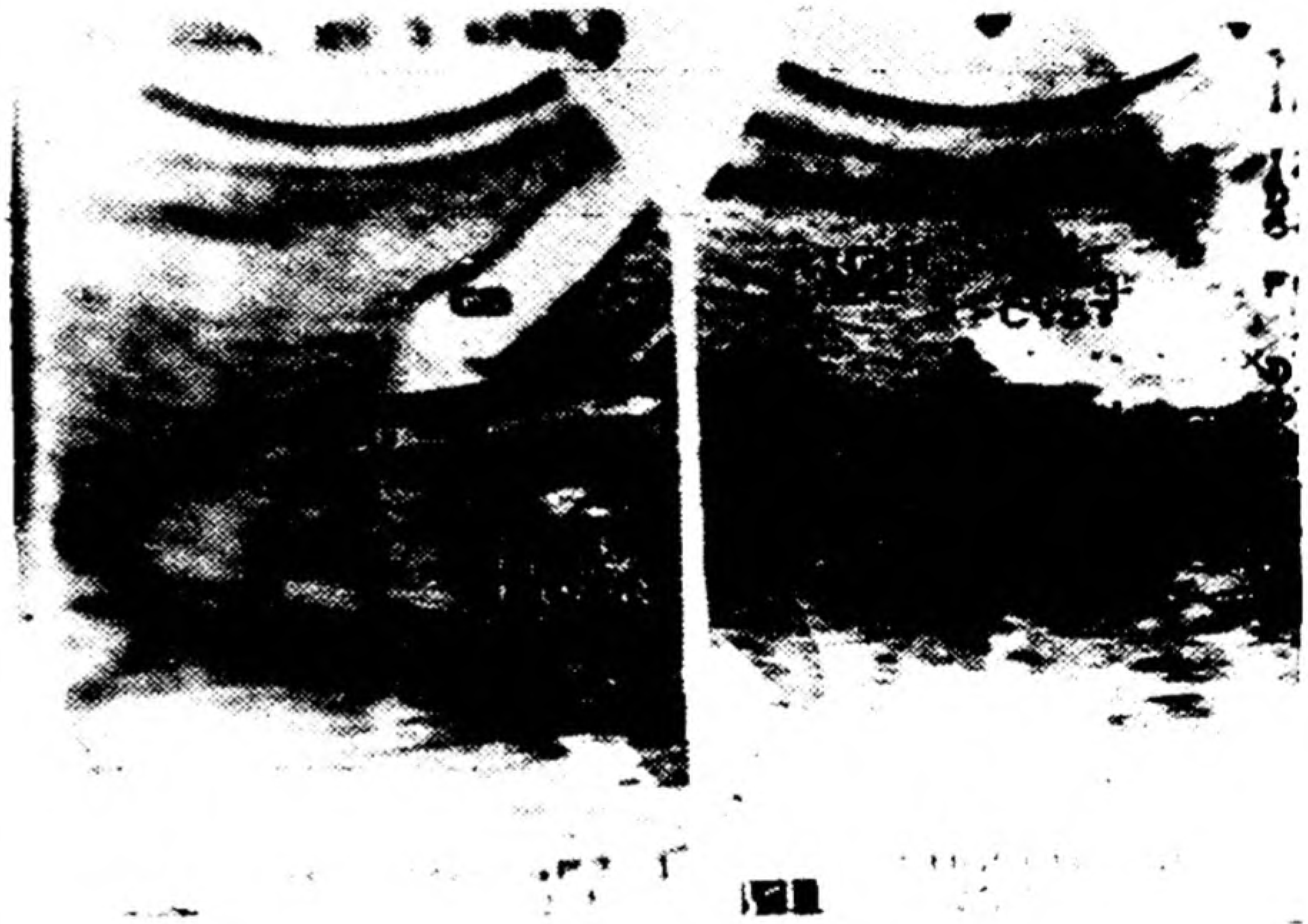

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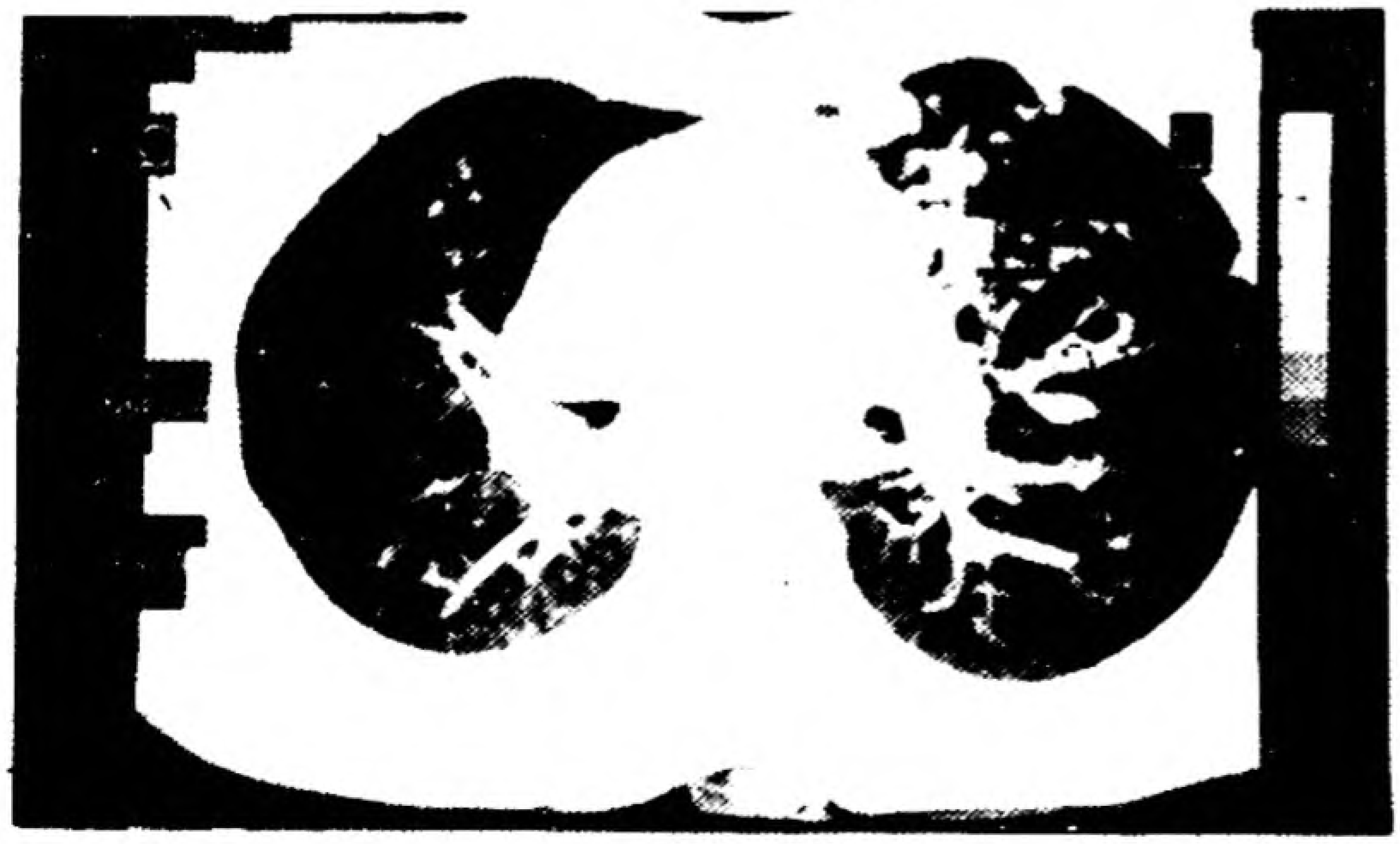

Fig. V

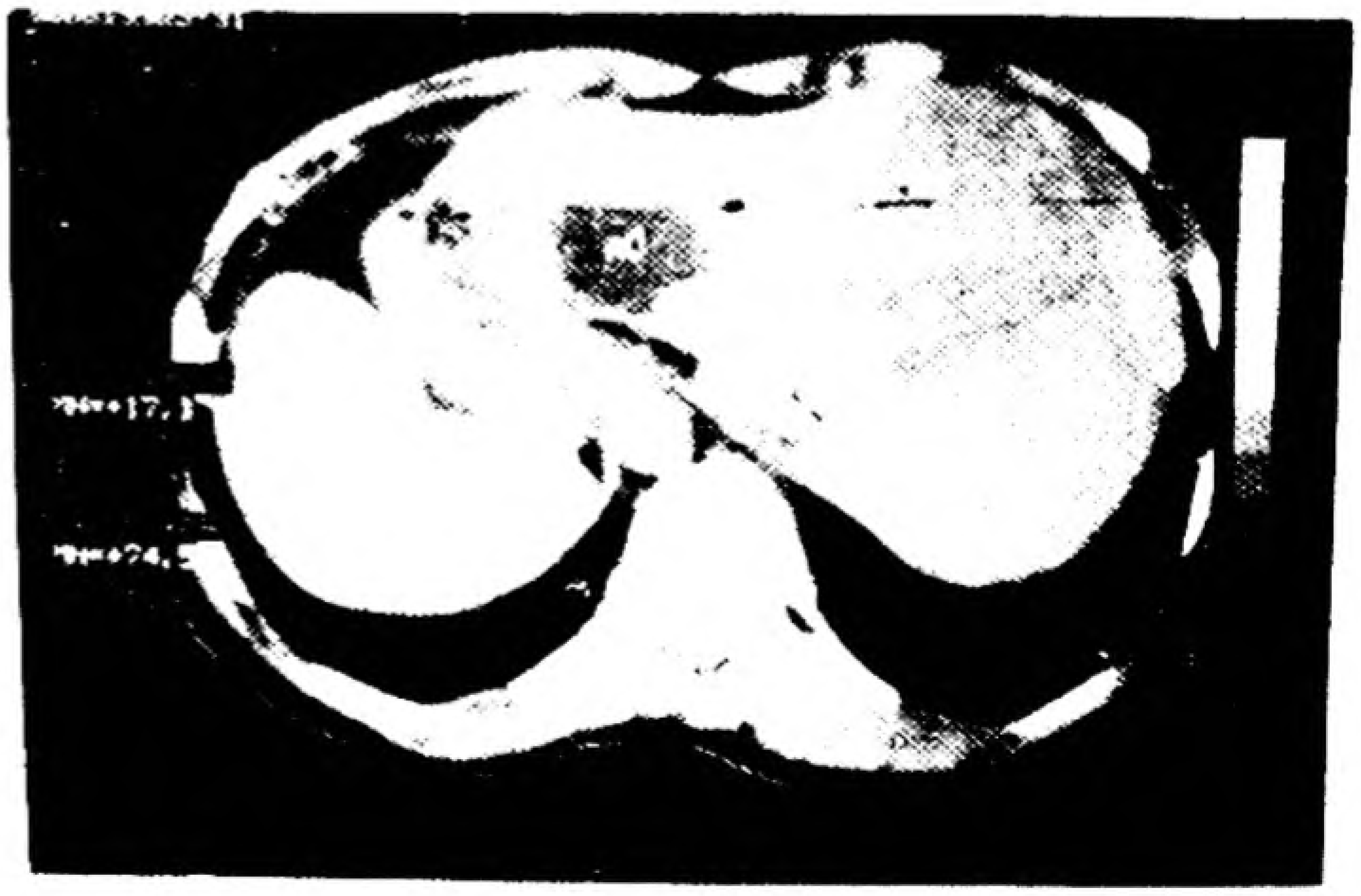

Fig. VI

E.C.G. (Fig.I) : Showed normal sinus rhythm with P and T waves inverted in lead I and negative major QRS deflection. Leads AVR and AVL were mirror images of normal. Precordial leads V_1 through V_6 revealed mirror image of normal. QRS axis was right.

X-Ray Chest (Fig-II) : Showed i/ fibrotic bands in left mid zome ii/ dextrocardia iii/ right dome of diaphragm lower than left dome iv/ gastric bubble below right dome of diaphragm v/ aortic knuckle on right side.

X-ray paranasal air sinuses (Fig-III) : Showed left maxillary sinus opacity and right maxillary sinus haziness.

Ultrasonogram of abdomen (Fig-IV) : revealed i/ liver present on left side with an intrahepatic cyst in right lobe of liver (morphological left lobe) ii/ single right sided spleen iii/ descending aorta on right side of spine iv/ pancreatic head directed towards left side.

The Casonis's test and hydatic serology were inconclusive

CT scan of chest and abdomen (Fig-V): Revealed i/ bilateral dilatation and destruction of bronchi ii/ dextrocardia iii/ situs inversus iv/ cyst in anatomical left lobe of liver without contrast enhancement. The audiogram was normal.

Discussion :

Kartagener's syndrome (KS) is an autosomal recessive disorder affecting[1] in 68000 in west[5]. In the present case no family member was affected either fully or partially (so called form fruste of KS). Our patient had chronic sinusitis with nasal polyposis, bronchiectasis and situs inversus i,e, Kartagner's triad. Besides there was a cyst in anatomical left lobe of liver. Of various known associations of the disease are male sterility, due to defective sperm tails and hypolasia or agenesis of nasal sinuses[8]. Our patient was diagnosed as a case of Kartagner's syndrome with simple hepatic cyst. On review of literature a combination of KS and hepatic cyst is not reported.

Address for correspondence:
Dr. G.M. Malik M.D. FACG
P.Box 884, Srinagar 190001.
Kashmir India

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"Xeroderma Pigmentosa and Skin Cancers"

Sheikh A. Ahad*; Mohammad Latif;**
Zahoor Ahmad*; Naseema Chanda******

JK Practitioner 1996; 3:3 165-166

Xeroderma pigmentosa is a hereditary disorder. transmitted as an autosomal recessive in which pigmentary changes, atrophy, keratosis and other progressive hyperplastic changes in skin with multicentric basal cell and squamous cell carcinoma and less often malignant malanoma occur. The malignant change is clearly a secondary phenomenon. The disease was first described by Hebra and Kaposi in 1893.

The incidence of xeroderma pigmentosa has been estimated at between 1:65000 and 1:100,000 of the population.

The histological changes in a fully developed case in the atrophic stage closely resemble those of senile skin. Irregular proliferation of retepegs heavily laden with pigment is a distinctive histological feature. Electron microscopy conducted by Rashud (1969) shows shrinkage of keratinocytes and their nuclei, widening of intracellular spaces, fragmentation of dermosomes and polymorphism of malanosomes, which are present in an increased number.

CLINICAL FEATURES:

Clinically skin is normal at birth and the symptoms of the disease develop in the early childhood in 75 percent of the cases (Hefnavi 1962) and most of the cases have reached tumour stage before the age of 20 years. Freckling and increased dryness on light exposed surfaces are commonly the earliest manifestations. Face and the hands are involved first.

Telangiectases and angiomata on unexposed skin and buccal mucous membrane have been reported (Goldman 1961), though these features are common on exposed skin. The disease is usually fatal and 75 per cent of the cases die before the age of 20 years.

The below mentioned family is reported because of the rarity of the disease and some of the associated electrocardiological findings and malignancies found in these patients and the absence of such reports in the literature.

CASE NO: 1

A.R., a well built male of 14 years of age, presented in the ENT clinic of S.M.H.S. Hospital, Government Medical College, Srinagar on June 1976, with the history of an ulcer on the right side of the ala of nose and a small nodule on the right eyebrow of one year duration. The patient also had pigmentation of the face and hands since early childhood. A throrough examination of the patient with a dermatological consultation and biopsy made it possible to label this patient as a case of xeroderma pigmentosa with basal cell carcinoma of the face. The patient was treated by surgical excision and alaplasty, After discharge, the patient was lost to follow up for two years and after this period presented with a recurrence of the ulcer at the eye brow and a mass which was infilterating the right eye ball with erosion of the dorsum of nose. Surgical excision of the mass with exenteration of the eye ball was carried out as the mass was now a squamous cell carcinoma. Post-operatively the patient received radiotherapy. An artificial eye was implanted for cosmetic reasons. The patient did well for 5 years when in 1983 July he found a swelling in the left side of cheek and an ulcer over the dorsum of the nose. Patient reported after three months of the onset of these fresh lesions and when he was examined he had squamous cell carcinoma of the maxillary antrum with infilteration into the orbit and involvement of the skin. patient was ,managed by giving a combination of Cryotherapy. radiotherapy and chemotherapy (Methotrexate, Blecomycin, Predinisolone). A marked improvement was observed clinically and patient free of from malignancy clinically at present after one year of follow-up but is left with cosmetic disfigurement (Fig. I B). which may be dealth with later on.

During the stay in the hospital all possible investigatyions were carried out to rule out any associated problems, all the radiological and biochemical studies were within normal limits except his electrocardiography which revealed a right bundle branch block.

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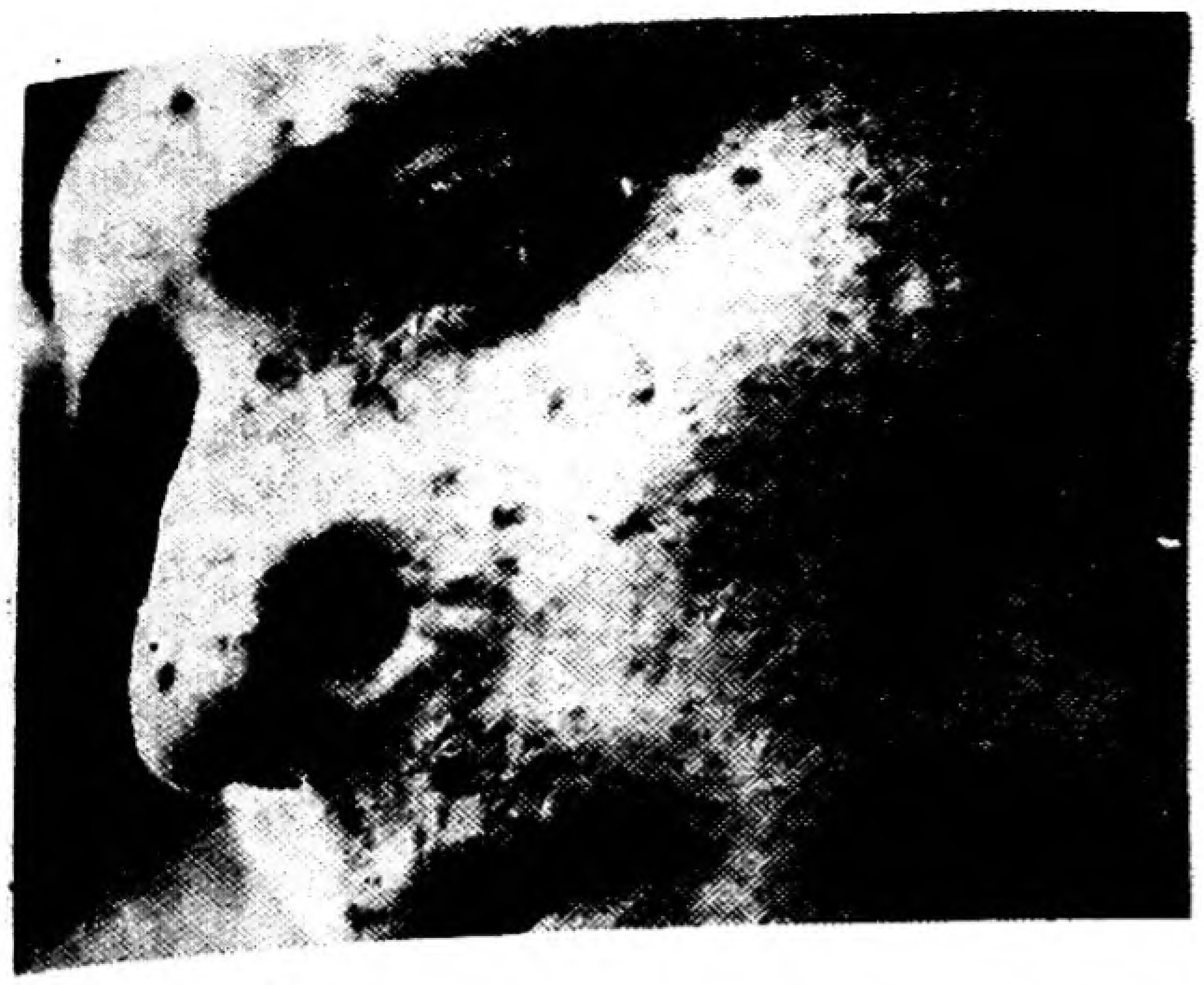
* Professor and Head
**Assistant Professor
*** X-Resident
****Demonstrator (Pathology)
Department of Otorhinolaryngology (ENT)
Govt. Medical College and Associated
Hospitals. Srinagar.



CASE NO: 2

A.B., of 10 years of age is the paternal cousin of case 1 and their family had consangunous marriage. This case had xeroderma pigmentosa with 5 cm diameter basal cell carcinoma on the dorsum of the nose. Patient was treated with cryotherapy and advised to avoid the sunlight. On investigations interestingly this patient also showed right bundle branch block.

CASE NO: 3

A young boy of 5 years of age the brother of Case No. 2 had xeroderma pigmentosa. This case is being watched for development of any new lesion. Electrocardiogram of this patient was normal.

DISCUSSION:

Malignant disease of the skin especially basal cell carcinoma are fairly common with xeroderma pigmentosa, which in itself is a rare entity, but the way our case have presented is rather an unusual feature of the disease. Our first case got different types of lesion and these were found in crops. The combination treatment in very advanced cases is encouraging. The conduction defects found in these patients on electrocardiography has not been reported in the literature, although the sudden deaths with anaesthesia in these cases and in cases of basal cell nevus syndrome have been reported. (Graeme et al). To derive any conclusion from these small number of cases will not be genuine but may prove a stimulus for all of us to investigate these cases fully whenever we get an opportunity to deal with such a case.

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Supraglottitis During Pregnancy

Parvez A. Mir M.D.*

JK Practitioner 1996; 3:3 167-168

History:

A 29 year-old, 12 weeks pregnant lady presented to the Emergency Room with a history of 2 days of a severe sore throat and painful swallowing. She had presented to a private physician one day earlier and had been given oral ampicillin for "pharyngitis".The following morning, the pain on swallowing liquids and solid food - worsened and prevented her from taking the prescribed antibiotic. She denied the presence of fever, cough, shortness of breath, myalgia, and a stiff neck as well as any previous health problems. She was noted to have a muffled voice.

Physicial examination:

Temperature: 101 F, Pulse 100/minute Respiratory rate 26/minute. Blood pressure 130/74 mm Hg. General condition : anxious, reclined, stridor present. Accessory ,muscles of respiration were being used. Skin normal Neck no masses. Chest stridor present,. heart; normal, tachycardia present. Abdomen normal

Laboratory findings:

Hematocrit:39%. While blood cell count; 14,000/Ul with 78% neutrophils, 20% lymphocytes, and 5% bands, Rapid screening for streptococal antigen: negative, Arterial blood gas revealed Ph: 7.49, PO2: 82 and PCO2 :27 on room air

Course:

Due to moderately severe stridor, and use of accessory muscles and impending muscle fatigue, a decision to perform and endotracheal intubation immediately was made. Direct laryngoscopy during intubation revealed erythema and swelling of the supraglottic structures. Patient was started on intravenous hydrocortisone and cefuroxime after all appropriate cultures were obtained. Patient showed a significant improvement and was weaned off the respirator without any fetal or maternal harm. Antibiotics were continued for a total of 7 days and steroids were tapered off after three days. patient was discharged on day 6 hospitalozation and continued the pregnancy without any further complications.

Diagnosis:

The incidence of acute epiglottitis in children has decreased markedly since the introduction of effective vaccines against Hemophilus influenzae type b in the U.S. In 1980 the incidence of pediatric epiglottitis was 3.5 cases per 100,000 population per year, by 1990, it had declined to 0.6 cases per 100,000 per year. However the incidence of epiglottitis remained relatively constant at 1.8 cases per 100,000 per year. During pregnancy special attention to airway maintenance should be paid to avoid maternal and fetal hypoxia.

Clinical manifestations:

The term epiglottitis is a misnomer for this condition in adults. Only 13% of adults over the age of 18 years have inflammation confined to the epiglottis. Most have associated erythema and swelling of the tongue, pharynx, uvula, arytenoepiglottic folds, and false vocal cords. Thus in adults the disorder is more appropriately termed supraglottitis. Traditionally, H. Influenzae type b has been the most common cause of supraglottis in adults. However recent series indicate that the prevalence of such cases has decreased. One reason may be the diminishing reservoir of H, influenzae in the pediatric population as a result of the effectiveness of existing vaccines.

Several pathogens that may be responsible for supraglottitis include Staphylococcus aureus, group A B- hemolytic streptococci, and Streptococcus pneumoniae. In addition, supraglottitis that is caused by Candida infection has been seen recently in patient with the acquired immunodeficiency syndrome.

Among adults with supraglottitis, the mean age at presentation is 45 years, but the disease has been reported in patients from 18 to 85 years of age. The male to female ratio is 1.8:1 Although the disease occurs slightly more frequently in the summer months, than at other times of the year, Few patients with supraglottitis have underlying chronic medical conditions, hence any adult is at risk for the disorder. The predominant symptom of supraglottitis are sore-throat and odynophagia, muffled voice, pharyngitis, fever and chills and drooling are seen less frequent. Stridor is seen in 12% patients only.

As a rule all adults with severe sore-throat and pain on swallowing should undergo an evaluation

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*Attending physician pulmonary
& critical care medicine
Wyckoff Heights Medical Center New York

for supraglottitis regardless of the pharyngeal findings. A muffled voice. drooling or stridor accelerates the urgency of the evaluation.

Indirect laryngosscopy is the preferred diagnostic examination for patients with suspected supraglottitis. Typical findings include erythema and swelling of the supraglottic structures. Adults tolerate this procedure well. In the absence of laryngoscopy. a lateral neck soft - tissue roentgenograms may demonstrate swelling of the supraglottic region. The sensitivity of soft-tissue roentgenography is lower than of indirect laryngoscopy. Fibreoptic rhinolaryngoscopy is preferred when available as it allows a detailed examination of the larynx.

Throat cultures were negative in this patient. as they frequently are in about 20% of cases. Prior antibiotic therapy may be responsible for negative cultures. Blood cultues are positive in only 15% of cases.

Treatment:

Most adults with supraglottitis can be treated medically in the intensive care unit. Patients who need to sit erect to maintain ventilation or exhibit stridor require endotracheal intubation and ventilatory support. A translaryngeal endotracheal tube is preferred. If difficulty is encountered in intubating the patient. cricothyroidotomy should be performed by an expert. Most patients can be extubated within 48 hours. General guidelines for extubation:

Improvement in general condition.

Visualization of the glottis directly with a Fibreoptic laryngoscope.

Finding that an air-leak exists around the endotracheal tube when cuff is deflated during spontaneous respirations.

Antibiotics:

Drug therapy for supraglottitis should include a 7-10 day course of an antibiotic with activity against H.influenza and other common pathogens. A second or third - generation cephalosporin like cefuroxime is the preferred agent. Cortiscosteroids reduce soft-tissue edema. but no data indicate that their use reduces the need for intubation or accelerates recovery. Most patients are discharged within four days of initiating parenteral antibiotic therapy.

Chemoprophylaxis:

Rifampin should be used in children younger than 4 years-old when exposed to an adult with epiglottitis regardless of their immunization status.

Complications:

Noncardiogenic pulmonary edema due to airway obstruction

Epiglottic ulceration or abscess

Spreading infection with mediastinitis and intrathoracic empyema

Patient's course:

After discharge from the hospital patient did well and both the fetal and maternal outcome was good.

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Short Case Report with Clinical Review

Extreme Metabolic Alkalosis and its Management: A Review of Two Cases

R. Bashir*; T. Mir**; F. Khan***

JK Practitioner 1996; 3:3 169-172

Metabolic alkalosis has been reported to be the most common acid-base disturbance in hospitalized patients. Significant mortality and morbidity can be seen in cases of severe metabolic alkalosis, including dysrhythmias, respiratory failure, seizures and coma. This, along with its potential reversibility with simple measures, has made it a subject of intense interest to acid-base physiologists and to clinicians over the last several decades. We report two cases of extreme metabolic alkalosis secondary to volume depletion that responded to aggressive management without sequence.

Case 1:

A 28- year-old male with a history of heroin and alcohol abuse in the past was admitted with severe vomiting and diarrhea. Patient had an extreme electrolyte disturbance with sodium $(Na)^+$ 113mg/l, potassium $(K)^+$ 3.7 mg/l, chloride $(Cl)^-$ 49 mg/l, bicarbonate (HCO_3) 49 mg/l, glucose (G) of 1360 mgm/d. and blood urea nitrogen (BUN) of 83 mgm/dl. Arterial blood gas (ABG) revealed a pH of 7.6, $PaCO_2$ of 51, and a normal PaO_2. Urine Na was less than 10 and urine chloride less than 15. Patient was diagnosed to be in hyperosmolar nonketotic state. Patient was aggressively hydrated with normal saline and received insulin infusion which corrected the metabolic abnormalities, and patient was discharged.

Case 2:

A 64-year old Arab female with a past medical history of renal insufficiency, mesenteric artery thrombosis, status post ileostomy, presented with dizziness. Physical exam revealed dehydration and orthostasis. Radiological Studies were normal. Electrocardiogram revealed left-axis deviation with a prolonged QT interval. Lab data showed Na^+ 127 mEq/l, K^+ 2.6 mEq/l, Cl^- 64 mEq/l, glucose 232 mg/dl, and HCO_3- 47 mEq/l. BUN and creatinine (Cr) were 75 and 4.3 mgm/dl, respectively. Ileostomy output analysis showed Na^+ 145 mEq/l, K^+ 6.4 mEq/L and Cl^- 136 mEq/L. Urinary Na^+ and Cl^- were 30 mEq/L and less than 15 mEq/l respectively. Arterial blood gas showed pH of 7.53, PO_2 of 59 mmHg and pCO_2 of 51.4 mmHg. Patient was hydrated with normal saline and required potassium chloride supplements. This regimen completely corrected the acid-base disturbance in the patient.

DISCUSSION

These two cases illustrate the need for prompt recognition of extreme metabolic alkalosis and its potential reversibility. The management of this disorder includes restoration of extracellular fluid volume with normal saline, along with potassium and chloride supplements.

KEY WORKS

Metabolic Alkalosis
Acid base balance
Extracellular fluid
Hypochloremia

INTRODUCTION

Most common acid-base disturbance in hospitalized patients (>50%)

Mortality: >45% - pH 7.54-7.65
>80% - pH>7.65

Definition: Arterial pH of >7.45, plasma bicarbonate >2.5 mmol/L, and compensatory hypoventilation.

CAUSES

Three Basic Mechanisms

A) Loss of H^+ ions from extracellular compartment.

1. Gastrointestinal tract: vomiting, nasogastric suction, colonic villous adenoma, antacids, cation exchange resins.
2. Kidneys: Diuretics (loop/thiazides), excess mineralocorticoids, post hypercapnic, decreased chloride intake, high dose carbenicillin.
3. Intracellular Shift: hypokalemia, refeeding.

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*Senior Resident
**Asstt. Professor
***Prof. & Head
Deptt. of Medicine
Nassau County Medical Center, East Meadowm NY; and State University at Stony Brook.

B) Chloride depletion and volume contraction loss of Cl- containing fluid with little or no bicarbonate. Diuretics. sweat losses in cystic fibrosis.

C) Bicarbonate retention: massive blood transfusion. NaHCO. therapy. milk-alkali syndrome.

CASE - I

A 28- year-old male with a history of heroin and alcohol use presented with vomiting. diarrhea. and confusion for 3 days. Physical exma:

Dehydration and orthostasis. mild epigastric tenderness

$(Na)^-$ 113.mEq/l. K^- 3.7.mEq/L. Cl^-<50mEqL. HCO_3^-49mEq/l, BUN 83mgm/dl. Creatinine 4.0 mgm/dl, Blood Sugar 1360 mgm/dl. Anion gap 14. pH 7.60. $PaCO_2$ 62 mmHg. PaO_2 132 mmHg, FiO 230%. Urine Lytes: Na^+ <10 mEq/d. K^+119 mEq/L. Cl^-<15mEq/l. Urine pH 5.0, Urine glucose >1000 mg/dl. Urine Ketones negative, Corrected calcium 7.8 mg/dl.

Treatment: hydration with normal saline. insulin drip, KCl supplements

Repeat ABG showed pH became 7.42 and HCO_3 29 mEq/L within 48 hrs.

CASE - 2

64-year-old Arab woman with h/o renal insufficiency. mesenteric artery thrombosis s/p ileostomy.

C/O dizziness for 1 day.

P.E. Pulse 83 bpm. BP 90/62mmHg.with orthostasis. Temp 98.4°F. and Respiratory Rate 16/min. Dehydrated.

Labs: Na^+127mEq/L. K^+ 2.6 mEq/L. Cl^-64mEq/L,HCO_3 47mEq/L. BUN of 75mgm/d. Creati nine 4.3 mgm/dl. Blood Glucose 232 mgm/dl. Anion gap 16. ileostomy output analysis:Na^+30 mEq/l. Cl^-26mEq/l. K^+89 mEq/l. pH7.53. Pao_2 59 mmHg. $PacO_2$ 51mmHg on room air

Treatment: hydration with normal saline. KCl supplements given.

This completely corrected the disorder in 24 hrs.

ADVERSE EFFECTS

A) Central Nervous System
   Confusion. obtundation. delirium
   Decreased seizure threshold

B) Neuromuscular
   Weakness. cramps. spasms. tetany

C) Cardiovascular
   Refractory ventricular and supraventricular arrhythmia's
   Enhanced digitalis toxicity
   Hypotension and decreased cardiac output

D) Pulmonary
   Alveolar hypoventilation and hypocapnia
   Atelectasis and V/Q mismatch and hypoxemia
   Hypokalemia induced respiratory muscle fatigue
   Leftward shift of oxyhemoglobin dissociation curve (Bohr effect) - impaired tissue oxygen delivery.

E) Metabolic:
   Decreased ionized calcium
   Increased lactate production
   Increased anion gap
   Hypokalemia and Hypophosphatemia

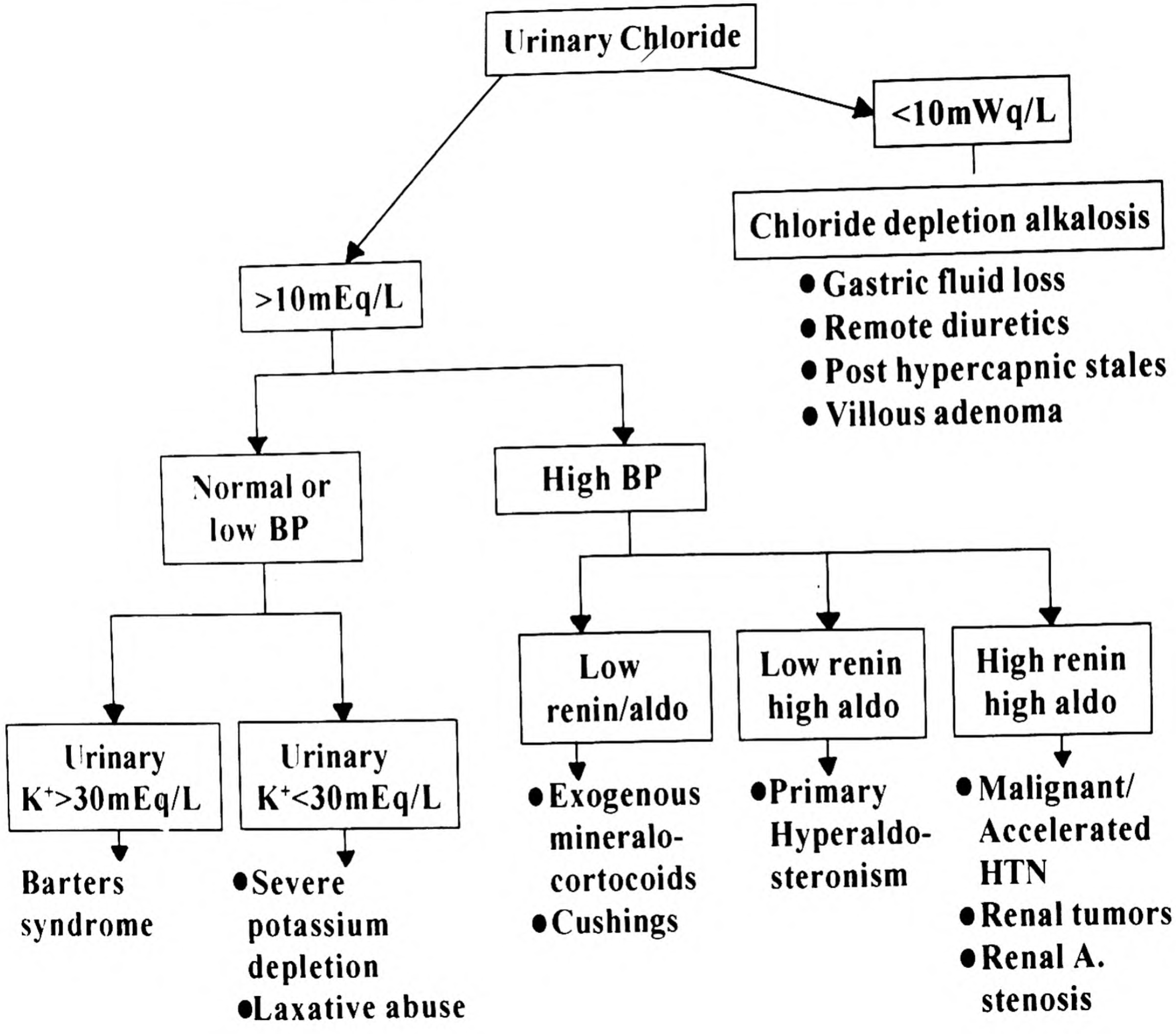
MANAGEMENT

Correction of inciting process
Restoration of volume and electrolyte depletion
Chloride deficit correction is essential
Calculation of chloride requirement:
= (0.2L/Kg) x (Body wt. in Kgs) x (desired Cl^- -measured Cl^-)
Initial dose should not exceed 4 mmol/Kg
Volume correction is usually done by normal saline.
Potassium supplements are commonly required in MA.
Renal function severely impaired. congestive heart failure. cardiac arrhythmias. hyperkalemia:
Acetzolamide (GFR is adequate)
HCL or NH_4 Cl. dialysis

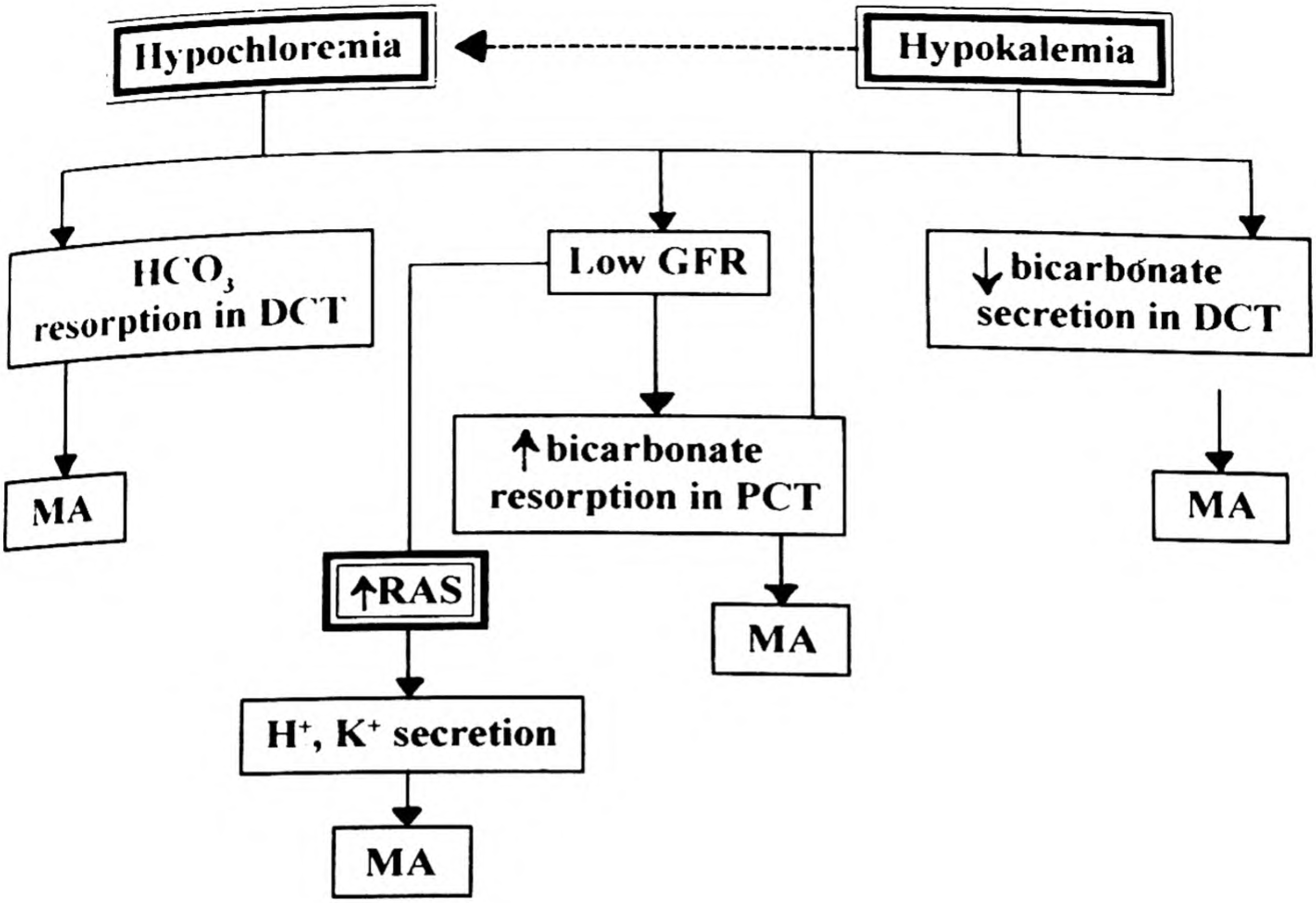
CONCLUSIONS

These two cases illustrate the need for prompt recognition of extreme metabolic alkalosis and it's potential reversibility. The management of this disorder includes chloride replacement alongwith restoration of extracellular fluid volume with normal saline. Potassium supplements are usually required

DIAGNOSIS OF METABOLIC ALKALOSIS


Urinary Chloride
>10mEq/L
<10mWq/L
Chloride depletion alkalosis
● Gastric fluid loss
● Remote diuretics
● Post hypercapnic stales
● Villous adenoma
Normal or low BP
High BP
Urinary K+>30mEq/L
Urinary K+<30mEq/L
Barters syndrome
●Severe potassium depletion
●Laxative abuse
Low renin/aldo
Low renin high aldo
High renin high aldo
●Exogenous mineralo-cortocoids
●Cushings
●Primary Hyperaldo-steronism
●Malignant/ Accelerated HTN
●Renal tumors
●Renal A. stenosis

Pathophysiology of Metabolic Alkalosis


Hypochloremia
Hypokalemia
HCO_3 resorption in DCT
Low GFR
↓bicarbonate secretion in DCT
MA
↑bicarbonate resorption in PCT
MA
↑RAS
MA
H^+, K^+ secretion
MA

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Doctor & Patient Relationship
"Holding a Hand is Often as Important as Examining One."

Lawrence H.Brandt*

JK Practitioner 1996; 3:3 173-177

The practice of medicine is in sorry shape today. Health care costs are oppressive and have skyrocketed to more than 900 billion dollars a year; we've made great advances in combating the AIDS epidemic, but the horrendous toll taken by this affliction continues and its cure is not just around the corner; patients are increasingly frustrated with an overburdened, bureaucratic health care system and are dissatisfied with their physicians. It is this latter sentiment that I wish to address before you today. Isn't it a paradox that at a time when medicine has made such great advances in unraveling the mysteries of human disease, that the tension, anger, and distrust between patients and their doctors have reached a pitch never before seen in our discipline? Perhaps not.

Patients change doctors frequently, and according to a Harris poll, when they do so, it is frequently because of concerns about doctor-patient communications. When asked why they changed their doctors, patients responded:

The doctors didn't spend enough time with me	51%
The doctor wasn't friendly	42%
The doctor didn't answer my questions honestly and completely	40%
The doctor didn't explain my problems understandably	30%
The doctor didn't treat me with respect	27%

Office visits to primary care physicians last a mean of 17 min, with 68.3% lasting less than 15 min (1). Unlike in architecture, however, the adage of Mies vander Rohe that "less is more" usually is counter productive when applied to interacting with patients. Moreover, physicians too readily assume the patient's first complaint is the most important, and label this the "chief complaint." However, Frankel and Beckman, at the University of Rochester, found there was no relationship between the order in which patients bring up their concerns and the medical significance of the stated problems, i.e., there was no evidence to support the notion nor validity of a "chiefship" among complaints (2). When given the opportunity to fully describe their spectrum of concerns, patients offer three complaints (3). Since there are data that suggest the first stated complaint is not necessarily the most important, premature interruption by the physician may prevent the expression of critical concerns (2). A major concern expressed by physicians about encouraging patient participation and open-endedness in the medical interview is that, given the opportunity, patients will talk forever. In their study, Beckman and Frankel found that when a group of 74 patients were asked, "what problems are you having?" none took more than 150 s to respond (3). Most patients, however, are not allowed to get that far. One of the most common, though unrecognized problems when doctors and patients talk, is that the doctor continually interrupts the patient, disrupting their attempts to complete a thought. In Beckman and Frankel's study of medical interviews between internists and their patients, 51 of the patients were interrupted by their physicians an average of 16 s after beginning to explain what was bothering them (2). The nature of the interruptions varied, but generally fell into four groups: closed-ended questions (46%): Doctor: "Have you had headaches before?" Answer: "No"; recompleters, in which an utterance restates the content or sense of content of the patient's previous statement (35%): Patient: "I'm out of breath." Doctor: "You're out of breath?;" elaborators, used to encourage the patient to continue speaking (10%): Patient: "I've been quite nervous lately." Doctor: "Tell me about this nervousness"; and statements pertaining to the patient's previous statement (10%): Patient: "I've been having chest pain." Doctor: "That sounds serious."

As a result of ineffective communication about patients' problems, in 50% of visits, the doctor and the patient do not even agree on the nature of the presenting complaint (4); whereas in a study by Burack and Carpenter, the patient's chief complaint agreed with the physician's determination of the patient's primary problem in 76% of somatic but only 6% of psychosocial problems (5). Control in the medical encounter is exerted principally by the use of questions. Although this style is an efficient

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*Montefiore Medical Center,
Bronx, New York

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means of establishing cause and effect, it severly limits the doctor's ability to obtain other crucial information and prevents the physician from creating an environment of caring.

When doctors dominate the medical interview, patients do less well than when control is shared; currently, however, when the patient does exert control, it completes with the traditional role of the physician and causes stress for both parties. According to a 1989 report by Drs. Sheldon Greenfield and Sherry Kaplan (6) at New England Medical Center, even a 20-min coaching session during which patients' records were reviewed with them, they were helped to think of questions they might want to ask and to evaluate their thoughts on medical decisions made during the office visit, translated into improved blood sugars, lowered blood pressures, and improved functional status, compared with the outcomes of patients who received information alone. Similarly, Roter (7) has shown that both patient satisfaction and adherence were positively related to an intervention designed to teach patients to ask more questions. Studies show that patients who clearly understand the need for certain medications and who feel their doctor is caring and supportive, are more likely to get better (4,6,7). Concordance between a patient and a doctor in terms of identifying the nature and seriousness of the clinical problem correlates with its resolution or improvement (4), whereas greater participation by the patient in the encounter improves outcome of therapy (6), satisfaction and compliance (7).

What is the best means of interacting with our patients so that we learn what is really bothering them, ensure that they get care that is both needed and in concert with their wishes, and also have them take away not only a sense of security that their physician is knowledgeable and concerned, but also empathetic and compassionate?

Compassion is truly a keystone of the relationship between doctor and patient. Even the words compassion and patient are inextricably linked, for both are derived from the latin, patior, meaning to suffer. Although all patients bear the burden of suffering to some degree, the truly compassionate physician is one who relieves suffering. Paracelsus viewed compassion as the doctor's schoolmaster, but a healing relationship is a balanced one of caring, compassion, curiosity, and competence, blended with hope, and two other well-springs of virtue, humility and humour (8). Thomas (9) has maintained that "touching (is our) real professional secret ... the oldest and most effective act of doctors." He cautions further: "The close-up, reassuring, warm touch of the physician, the comfort and concern ... are disappearing from the practice of medicine, and this may turn out to be too great a loss for the doctor as well as the patient." To do it right has never been easy; it requires the best of doctors, the best of friends. Once lost, even if for one generation, it may be too difficult a task to bring back again (9). We must learn again and teach future generations of physicians that holding a hand is sometimes more important than examining one; that when we prescribe pills, tablets, capsule, and caplets, we must do so with equal doses of compassion, support, and understanding. Rosen (8) has suggested that the "soft" art of healing, an interesting word derived from the old English word, haelen, meaning to make whole, is complemented by a "hard" or scientific basis for the doctor-patient relationship. He finds evidence for this in the studies of Jane Goodall and Dian Fossey, who worked with chimpanzees and gorillas, respectively, and observed them exhibiting healing behavior: removing splinters, giving dental care, draining abscesses, and providing the most comforting of healing gestures, touching. Rosen contends that the doctor-patient relationship is founded on a biology of acceptance and trust, which is rooted in archetypes, or inherited modes of psychic functioning.

There have been many studies of what constitutes the best form of doctor-patient relationship and as one might expect, no single one is best in all circumstances, Clearly, over the past two decades, there has been a struggle over the patient's role in medical decision making, characterized as a conflict between autonomy and health, the values of the patient and the values of the physician (10). Emanuel and Emanuel (10) described four models of the doctor-patient interaction. emphasizing the various goals, obligations, and values of the patient and the physician, as well as the concept of patient autonomy. In the traditional, paternalistic model, the doctor-patient relationship serves to enable the physician to discern what is in the patient's best interest with limited patient participation. The physician presents the patient with selected information that will encourage the patient to agree to whatever it is that the physician considers best. This model assumes shared objective criteria for determining what is best. The goal of this model is the health and well-being of the patient and the physician acts as the patient's guardian. Thus if an 82-yr-old man with advanced parkinson's disease was bought to my office by his family because he had some rectal bleeding, but he was not anemic, and I told them it was not in their father's best interest to be subjected to any further testing, that would be paternalistic.

In the informative model, the physician provides the patient with all relevant information for the patient to select the medical intervention desired. The physician then executes the chosen intervention. The physician's obligation is to provide all the facts, whereas the patient's values-not those of the doctor-determine what therapy is to be given. In the case above, if I were acting as an interpretive physician, I would explain to the patient the survial of similarly affected persons and discuss the various causes of hematochezia in one his age; he would then instruct me whether he wished to have the problem pursued further.

In the interpretive model, the aim of the doctor-patient interaction is to provide certain information to the patient, elucidate the patient's values, determine what is actually wanted, and help select the intervention that realizes these values. The patient's values are not fixed and may not even be known to the patient. In this model, the physician acts as a counselor, helping the patient to understand their values. Thus, for my octogenarian exemplar, knowing from previous discussions that he does not wish to have major interventions done to prolong his life, I might recommend colonoscopy, after explaining that most of the major causes of rectal bleeding in a man his age can be, in large measure, controlled by coloscopic therapy, thereby probably enabling him to attend his granddaughter's wedding in 4 months.

In the deliberative model, the aim of the interaction is to help the patient determine the most correct, i.e moral, worthy health-related values that can be achieved in a given clinical situation. The physician acts as a friend helping to determine the best course of action. knowing that my patient has been a beacon of courage for all Parkinsonian's in the region, I might encourage him to pursue an evaluation and optimistic attitude so similarly afflicted persons won't lose hope. Clearly, each of the above models are not appropriate in all circumstances. For example, in an emergency, where a delay in treatment might irreversibly hurt the patient, the paternalistic model is correct. In recent decades, there has a shift toward greater patient autonomy, conceived as patient choice and control over medical decisions. Illustrative are: patient's rights statements, living will and right-to-die laws, doctrine of informed consent, and rules regarding human experimentation. Patient-oriented standards, in which the duty of the physician is to provide appropriate medical facts empower the patient to use his/her values to determine what intervention should be implemented.

As Spiro has said, once physicians cultivated their unique gift, to witness the human condition (11). They had the time to listen since there was so little else they could do. Living alongside their patients, whose lives they shared, they often understood them better. Now they live apart and doctors try to be as objective, perhaps distant, as possible. Although society clamors for closer relationships between physicians and their patients, external forces demand rapid, "bare-bones" medicine with no rewards for careful listening. A quotation from Tinsley Harrison, offered in 1944, is as appropriate now as it was then: " The present day tendency toward a 5-min history followed by a 5-day barrage of special tests in the hope that the diagnostic rabbit may suddenly appear from the laboratory that is professionally unwise and economically unsound." Should the doctor with limited time listen to how frightened the patient became when passing bright red blood with his or her bowel movement or perform a colonoscopy and perhaps resolve the problem? In a time long age, the patient with an intractable peptic ulcer had to find out what they were all about to get relief, perhaps change their lifestyle and their ambitions. Now, relief is just a swallow away; you take an H2 blocker or a proton pumpp inhibitor and you are well. Do such people learn about themselves and their lives? Are they better people? Does it matter? Can physician who bear witness offer hope for a better understanding of the human condition (11)?

We are now in the era of patient consumerism, practicing so-called Medicine of Strangers, rather than the Medicine of Friends; in the former, regulations and guidelines typify our doctor-patient relationship whereas in the latter, trust and friendship are dominant (12, 13) But the relationship between doctors and their patients has always been influenced by external forces: social, political, legal and economic. Unfortunately, in recent years, this relationship has been most altered and, in my opinion, for the worse, by health care costs.

In 1982, Jonsen et al. (14) developed a four-part approach to medical decision-making.

1. Medical indications
2. Patient preferences
3. Quality of life
4. External factors

The first, medical indications, is the physician's domain, wherein, after listening to the patient's complaint, the doctor makes a series of diagnostic and therapeutic recommendations. The second, patient preferences, emphasizes the patient's rights to determine what course of action is taken and encompasses the legal doctrine of informed consent. Conflicts between the two realms of the doctor and patient are, and usually result from misunderstanding, lack of commumication or trust, or a fundamental disagreement about the goals of the clinical encounter. As Siegler points out (12), from 1960 to 1980, medical indications and patient preferences determined the outcome; doctors hesitated to make decisions based on assessment by an external party of how good or bad somebody else's quality of life is, after which the external party would decide on the allowable limits of treatment or external factors, such as cost of services or legal implications of the care rendered. Over the past few years, however, there has been an increasing tendency to apply such external considerations to our decision-making process, thereby further weakening the already compromised autonomy of both the patient and the physician.

Siegler (12) has gone on to describe three ages in the evolution of medicine: the first stage, that of the physician, lasting from 500 B.C. to approximately 1965, in which medical achievements were modest by modern standards and the doctor's clinical judgemtn meaningful; the second, the age of the patient, lasted only form 1965 to October 1, 1983, the day that DRGs were born; and the third, which we are currently witnessing, the age of the payer. During the first two stages, the doctor-patient relationship was insulated from external interference, whereas in the age of the payer, the power of decision-making has shifted from those who provide medical care or those who receive that care, to those who pay for it. Similarly, success or failure of a given who is not judged by the degree of wellness or recovery of the patient, but rather by the cost of the clinical encounter. Under the weight of economic concerns and the increasing political pressures to do something about them, we are witnessing profound changes in the doctor-patient relationship, changes by which Siegler has predicted the decision-making will move to more external considerations, changes in which the decision-making authority “so reluctantly relinquished by physicians during the past 20 years and so enthusiastically embraced by patients, will pass from both of them to third parties” (12).

Respect for patient autonomy and self-determination as well as the negotiations, accommodation, and bonding that typigy a mature relationship between doctor and patient take time to develop, but time is a priority commodity in this brave new world of health care. Reimbursement is the lingua franca in the current and proposed systems of health care delivery, and it is likely to limit the time spent in building these relationships. Moreover, patient autonomy may become a one-way street, in which their decisions to stop treatment will be respected, but not their wishes to continue it-especially when care will not be reimbursed for therapy that has been deemed futile, the so-called futlity standard. Managed care and guidelines will limit the autonomy of both the patient and the physician, and guidelines that once served as an aid to decision-making will now dictate it. As the structure of a National Health System emerges from the shadows and as its multiple levels of bureaucracy become releato us, as physicians become more concerned with length of stay, DRGs and RVUs, and as terms such as “health provider” and “health consumer” - or even worse' “ client,” are increasingly used, the specialness of the relationship between the doctor and patient is threatened by a kind of leveling an commercialism which does not serve commitment well (15). Doctors have patients, lawyers and bureaucrats have clients; client from the Latin, clients, meaning a vassal of a feudal lord for whom services such as protection were rendered in return for payment.

We are, in our relationships with our patients, moving from one of accommodation to one of absolutism; the former is a kindly address of particular concerns based heavily on communication, discussion, and negotiation between doctor and patient (16), each respecting the other's individualism; the latter is based on a set of immutable rules. As pointed out by Dr. Howard Spiro, doctors yearn for absolutes-even in moral reasoning; for rules can be memorized, parameters established. Unfortunately, the ambiguities of a given patient's particular circumstances and the realities of clinical practice are best recognized in a society that tolerates or even rewards outliers, not one that penalizes them. We should, as the medical ethicist, Albert Jonsen, wrote, “ learn to accommodate the hard edges to the blurred outline”(16).

Telling the truth has been trumpeted as a prime virtue over the past 20 years and has replaced the paternalistic regard for what the physician thought was in the patient's best interest (17). Another Spiroism is that doctors are trained to truth like dogs to fetch; but whose truth (18)? The emotional truth of an elderly loved one dying in the ICU cannot be equated with the practical truths of bed availability in an overcrowded hospital system. It would be simple if ethics, the practical arm of philosophy, could be absented from the specifics of a particular case, but ethical decision-making does not lend itself to the algorithms of clinical decision-making.

Twenty-five hundred years ago, in The Laws, Plato described two types of doctor-patient relationships (19). In one type, which he called slave medicine, slave physicians took care of slave patients. The slave physician "never gives him any account of his complaints, nor asks him for any; he gives him some empirical injunction with an air of finished knowledge in the brusque fashion of a dictator and then is off in hot haste to the next ailing slave..." In contrast is the "medicine befitting free men," in which the citizen physician "treats their disease by going into things thoroughly from the beginning in as scientific way and takes the patient and his family into confidence. Thus, he learns something from the sufferers... He does not give prescriptions until he has won the patient's support, and when he has done so, he steadily aims at producing complete restoration to health by persuading the sufferer into compliance."

In the United States at this time, we may be revisting history and heading toward the widespread practice of slave medicine. The best caution against this is to maintain the sancity of the doctor-patient relationship and to remember why we went into medicine in the first place: it was to help people. When we talk to our patients and, perhaps more important, when we listen and hear what they are trying to say, when we are caring, compassionate, and sensitive to their needs, we are helping them-helping them not only to feel better, but do better. You'll find, to your surprise, that you'll feel better too. As Francis Peabody wrote in 1982: " The good physician knows his patients through and through, and his knowledge is bought dearly. Time, sympathy, and understanding must be lavishly dispensed, but the reward is to be found in the personal bond that forms the greatest satisfaction in the practice of medicine. One of the essential qualities of the clinician is interest in humanity, for the secret in caring for the patient is in the care of the patient" (2)).

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"Islamic Teachings and Cancer Prevention"
Tobacco and Cancer

M.A. Albar, FRCP

ABSTRACT: It has been widely reported that 80 to 90 percent of all cancers are related to life style and environmental factors, which could be in some way preventable. Lung cancer is the most vivid example as it is evidently related to tobacco smoking. In the U.S.A. alone some 100,000 die annually because of lung cancer. At least 80 to 85 percent of the case are directly related to smoking. Active cigarette smoking is responsible for 400,000 deaths annually in U.S.A. while passive smoking kills 53,000. Worldwide tobacco smoking and chewing is responsible for 2.5 million deaths annually. All these catastrophic mortalities are preventable.

Tobacco smoking and chewing is also responsible for many other cancers e.g. cancer of the mouth, cheeks, tongue, lips pharynx, larynx and oesophagus. It is also a contributing factor in cancers of the bladder, kidney, prostate, cervix and stomach.

Alcohol consumption multiply the effects of tobacco smoking and chewing on cancers of the oral cavity, pharynx, larynx and oesophagus. Heavy drinking is related to cancer of the liver and colon.

Homosexuality, promiscuity and early sex with multiple partners is related to a surge of sexually transmitted diseases as well as increased incidence of cancers of the cervix uteri, vagina, penis and anal cancer. Cancer cervix is related to human papilloma virus, genital herpes and genital warts. Even the classical venereal diseases e.g. syphilis, granuloma venerium and lymphogranuloma inguinale are related to malignant change. HIV infection is causing AIDS which involves malignant disease e.g. Kaposi Sarcoma and increased malignancies.

Excessive exposure to solar radiation as in sunbathing, especially in fair skinned, is associated with increased incidence of all types of skin malignancies, e.g. squamous cell carcinoma, basal cell carcinoma and melanomas.

Islamic teaching prohibits smoking, alcohol drinking, homosexuality, promiscuity (premarital and extramarital sex) and sunbathing and nudeness. These teachings, if adhered to will curtail greatly the incidence of malignant disease. Circumcision of males will prevent cancer of the penis, and early marriage and lactation will markedly reduce cancer breast.

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Introduction

It is estimated that 80 to 90 percent of all cancer cases are related to environmental and life style influences.(1) The majority of human cancers are preventable.(2) Extrinsic factors include, beside chemicals, infections (viral and parasitic), nutritional and a variety of other factors determined wholly or partially by personal behavior. Though genetic factors and age affect cancer onset rates, it does not affect the conclusion that much human cancer is avoidable(2).

Islamic teachings which control human behavior and personal life style can dramatically influence the rate of incidence of many malignancies. Many cancers are related to tobacco smoking and chewing, alcohol drinking, homosexuality, promiscuity and excessive exposure to solar radiation as in sunbathing, which are prohibited in Islamic teachings. Adherence to these teachings will curtail the incidence of many malignancies and curb the spreading epidemic at least in Muslim popula-

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Consultant of Islamic Medicine
king Fahd Medical Research Centre,
King Abulaziz University, Jeddha (Saudi-Arabia)

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tions. which amount to one billion.

Dr. John Hill, a London Physician, reported an increase of lip cancer in pipe smokers in 1761 and Sir Percival Pott reported cancer of scrotum in chimney sweepers in 1777, which he attributed correctly to lodgement of soot in the rugose scrotal skin. This type of cancer was virtually eliminated by simple personal hygiene. (3) Later, many chemicals were proved to be carcinogenic and hence many cancers became avoidable by taking more care when dealing with these chemicals.Ionizing radiation is still an important hazard which could be prevented by meticulous care.

Tobacco and Cancers

In many countries cancer is the second most important cause of death. In U.S.A. and many developed countries, it accounts for 20 percent of all deaths. It is estimated that 6 million new cases of cancer occur annually worldwide, of which about a million cases are caused by tobacco smoking and chewing.(4) The death toll due to malignant disease in U.S.A. amounts to 400,00-450,000 annually of which 100,000 are due to lung cancer.(1,2) 85 to 90 percent of lung cancer, which is the most important cancer, is due to cigarette smoking.

Tobacco smoking, chewing and sniffing is the most important single factor in cancer causation, and it is estimated that 30-40 percent of all cancers are related to tobacco smoking, chewing or sniffing.(5) No other single agent has been examined in much detail, is more firmly established as a causal agent or is responsible for more cancer deaths than cigarette smoking.[6] The risk of cigarette smokers developing lung cancer increases with the number of cigarettes smoked, the duration of smoking , the time of onset and the type of smoking. Approximately one sixth of those who smoke two packs of cigarettes per day will eventually develop lung cancer. Those who smoke40 cigarettes per day are having a 25 fold increased risk compared to non smokers.[6-8] Cigarette smoking causes all of the major types of lung cancer including squamous cell carcinoma, adeno carcinoma, oat cell and large cell carcinoma. Cancer of the lung was a rare form of cancer at the beginning of this century, even in developed countries. As smoking increased dramatically after world war I among men, and among women after world war II, the incidence of cancer of the lung showed incessant increase until the seventies, after which it began its descent for men and it took another decade to show the same trend in women. Nevertheless, cancer of the lung is still the first killing cancer among men and women in many developed and developing countries.[7,9-13] The rate of lung cancer deaths has been dramatically increasing in many developing countries. In Hong Kong, the rates for women are now the highest in the world.(13) Lung cancer rates in chinese men e.g. 50.2 per 100,000 in Shanghal are higher than in many North American and European populations. It is the leading cause of cancer mortality among males in Bulgaria, Cuba, Czechoslovakia, Egypt, Greece, Hong Kong, Hungary, Israel, Philippines, Poland, Romania, Singapore, Thailand and Uruguary and Zimbabwe. The risk is particularly high among cigarette smokers, and a clear cut dose-response relationship has been confirmed. The risk is greater among those who started smoking at a young age and those who smoke high yield.(2,14-16)

Laryngeal cancer is the second cancer caused by cigarette smoking, but the total number of cases is smaller than lung cancer and the survival is much better.(6) Cigarette smokers are five times more likely to develop cancer of oral cavity and the esophagus compared to non smokers.(6,7,8) There is synergism between alcohol & cigarette smoking in causing cancer of the larynx, esophagus and oral cavity.(6,17-21)

Cigarette smoking is also a contributing important factor in cancers of the bladder, kidney and pancreas.(6-8,14-16,22) Association between gastric cancer and smoking has been noted.(23-26)

Cigarette smoking has even been implicated in breast cancer, kidney, liver, cancer cervix uteri and many other cancers. (27,28,29) It was also related to childhood cancer as a result of prenatal exposure to parents smoking.(30)

Passive smoking was implicated in causing many cases of cancer.(31-33)

Smokeless Tobacco

Long term use of chewing tobacco or snuff has been linked to cancer of oral cavity, cheek, gums and oropharynx.(6) Oral cancer is one of the ten most common cancers in the world. In Bangladesh, India, Pakistan and Sri Lanka, it is the most common malignant disease and it accounts for a third of all cancers. More than 100,000 new cases occur annually is South and South East Asia.(13) The commonest cause for oral cancer is tobacco chewing, usually in the form of betel quid which consists of betel vine leaf (piper betel), areca nut , lime and tobacco. (13,34) Tobacco chewing is also widespread in parts of Yemen, Sudan and Southern province of Saudi Arabia. The so called "Shamma" is a tobacco plus lime and ash. It has been implicated in many cases of oral cancer in these areas.(35,36)

Smokeless tobacco was propagated in developed countries by tobacco companies after the decline of cigarette smoking there. The 39 World health Assembly in 1986 adopted Resolution WHA39 which declared that "the use of tobacco in all its forms is incompatible with the attainment of health for all by the year 2000.(37)The study group concluded that the use of smokeless tobacco caused cancers in humans, the evidence of casualty being strongest for cancers of the oral cavity. It also increased the risk of cancers of the nasal cavity, pharynx, exophagus, pancreas and urinary tract. Laboratory studies clearly supported the observations that smokeless tobacco caused a number of precancerous oral lesions.(37)

In conclusion, tobacco smoking, chewing and sniffing is related to cancers of the lung, oro-pharynx, larynx, esophagus, stomach, kidney, bladder, pancreas and many others. There is synergism between alcohol and cigarette smoking in causing cancer of the larynx, esophagus, oral cavity, liver and stomach.(6,17-21,29) Overall, tobacco consumption is responsible for 30-40 percent of total cancer mortality in many countries including U.S.A., Hong Kong, China, India, Bangladesh and many European countries.

Carcinogens in Tobacco Smoke

Tobacco smoke is an aerosol consisting of about 2000 different substances, 50 of which are already proven to be human carcinogens e.g. Benza pyrines, Benza anthracene, Benzo flouranthene, Benzene and other Benzyl derivatives, cadmium, chrysene, 5 methyl chrysene, methyl flouranthene and nitroso compounds.(38)

The smoke aerosol consisting of a gaseous and particulate phase, both of which contain many respiratory carcinogens, initiators of carcinogenic process and tumor promoters.(39)

There is ample clinical and laboratory data proving the association of cancer and tobacco smoke. Cigarette smoke also contains significant amounts of radioactive substances e.g thorium Th228, polonium-210 and radium- Rd226. These compounds lodge in the lungs where they constantly irradiate the nearby cells and hence facilitate malignant change.(38) Even the urine of cigarette smokers contains mutagenic substances for bacteria, and substances that cause changes in the chromosomes of human cells tissue culture.

Side stream smoke which is inhaled by the non smokers contains 50 fold greater concentration of nitrosamines than mainstream smoke. In one hour of breathing in a smoke filled room, a non smoker may inhale an amount of nitrosamines equivalent to the amount inhaled after having smoked 15 filter cigarettes.(38)

The available data confirms that: tobacco smoke contains multipotential carcinogens which produce different tumors at different sites. Laboratory data, the presence of radioactive substances in tobacco smoke and the presence of mutagenic substances in the urine of smokers all provide additional proofs that tobacco smoke is carcinogenic and is the most important substance related to human cancers and deaths.

Cessation of Smoking

The risk after cessation of smoking decreases dramatically. Light smokers approximate the risk of non smokers after 10 to 15 years of cessation of smoking, while heavy smokers have a residual two to three fold increase after cessation. (2,6) The mechanism of lung carcinogenises and smoking cessation has been extensively studied.(40)

Women and Smoking

Women started smoking long after men and hence there is a lag time of the incidence of cancer lung. The incidence of cancer lung has already fallen for men in most developed countries while it was still increasing for women early in the eighties. In 1984, 32 percent of the women smoked in Britain compared with 36 percent for men.(41) In 1961, 60 percent of the men were smoking in Britain while 40 percent of women smoked. The average women smoked half as many cigarettes as her male contemporary in 1950, while in the eighties they were almost smoking similar figures viz 16 for man and 14 for woman.(41)

During the second decade of anti-smoking campaign in Britain, smoking started to fall among women too, albeit slowly at first, but it is gaining momentum and accelerating.(42) Similar trends are found in all developed countries. In some countries e.g. Australia, smoking among young and middle aged women was rising in the early eighties.(5,43,44) The proportion of men who smoke was falling in 19 of 22 developed countries, while it was rising or stable in 11 countries for women.(42-44) In Austria, Germany, Italy, Japan, U.S.S.R., smoking among women was still rising in the early eighties.(42)

Cancer lung is the first killing cancer in many developed countries in men and women. It has already surpassed breast cancer. Non smoking wives of smoking husbands (passive smokers) are also inflicted with cancer lung,(6,31-33,37) and non smoking women are also affected at work places.

Smoking Trends in Developed and Developing Countries

Anti-Smoking campaigns have been launched in the developed countries in the last two decades with tremendous achievements. In Britain, 60 percent of men were smokers in 1961 (the year before the first report of the Royal College of Physician about smoking was published). By the time the second report was published in 1971, this figure had dropped to 47 percent and by 1984 only 36 percent were smoking. It is expected that by 1992 less than 25 percent of the adult males will be still smoking. The trend among women took another decade to show similar changes. From 1961 to 1971, the percentage remained static at 40 percent. By 1984, 32 percent of women in Britain smoked.(42,45)

Similar trends were observed in U.S.A., Europe and other developed countries About 30 million U.S. population and 10 million British persons have stopped smoking by mid-eighties. Cigarette sales have plummeted by an impressive 28 percent.(42) The trend is 5-10 percent loss annually.

This achievement has been confronted with a tragic promotion of cigarette marketing in the poor third world which is already suffering from serious health hazards. Consumption of tobacco in Third World has seen a horrendous increase. W.H.O. noted that tobacco related diseases are on the rise in developing countries. The rate of lung cancer has been steadily increasing in Hong Kong, China, India, Bangladesh, Sri Lanka, Pakistan and Philippines. Lung cancer is the leading cause of cancer mortality in China, Cuba, Egypt, Hong Kong, Israel, Philippines, Singapore, Thailand, Uruguay and Zimbabwe.(14)

All developing countries have shown a marked increase in tobacco smoking during the last two decades. In Senegal the percentage of men who smoke in urban areas has reached the unprecedented 80 percent. In Bangladesh 70 percent of the adult male population smoke. In Lagos 72 percent of the Faculty of Medicine male students were smoking.(46)

It is clear that the tobacco companies which manufacture 10 billion cigarettes daily are making up their losses in the developed countries by increasing their sales in the poor developing countries.

Tobacco is imported in Saudi Arabia and statistics from the chamber of Commerce show an unbelievable increase of tobacco imports from 4,575,267 kgs in 1972 to 27,026,836 kgs in 1977 which high rocketed in 1981 to 36,732,500 kgs,(47) an increase of 800 percent. Since then the increase is modest, nevertheless Saudi Arabia was the world's third leading importer of U.S. made cigarettes in 1984.(48) It is not amazing to find that lung cancer in Saudi Arabia has jumped by leaps and bounds. it was the 12th most common cancer in 1950-1961 study,(49) it became the third most common cancer in 1979-1984 study. (50) It is expected to be the leading cancer in the nineties as the effects of smoking are unfolding.

The unscrupulous tobacco companies have launched the most aggressive promotion campaigns in the third world where devious methods of corruption are used with government officials to allow promotion policies. In 1982, the head of the Malaysian parliament retired and went to work as a chairman of Rothman's, Malaysia's largest cigarette manufacturer.(48,51) Ethiopia imported 200 million expensive British cigarettes in 1984 when a large portion of its population were starving to death. In Bangladesh, smoking of five cigarettes daily robs the family of quarter of its food supply, which results in estimated 18,000 deaths among children annually.(53) Unfortunately, World Bank and Western Governments are co-operating with the seven giant tobacco companies (3 American, 3 British and one French). The World Bank has given Pakistan 60 million dollars in loans to raise tobacco and The United States Food for Peace Programs have spent 2 billion dollars in loans to developing countries for the purchase of U.S. tobacco and to erect joint venture tobacco projects and factories.(48,51) Tobacco needs curing with heat, which is obtained by burning wood. This results in deforesting 7 million acres annually, with its detrimental sequelae on the ecology. Tobacco growing needs a heavy use of pesticides. many of which are carcinogenic. In developing countries they are used without protective measures and end in many fatalities. Some of the imported pesticides are banned in the original manufacturing countries.

Advertising for tobacco has no limits in third world countries and many of the bands marketed contain high yield tar. Cigarettes smoked in China, India, Pakistan, Sri Lanka, Philippines etc, contain 21-33 mg tar and 1 mg nicotine.(13,46,48)

China consumes one third of the worlds production of cigarettes, while the other developing countries consume another third.(48,54) The Eastern and Western block combined consume the remaining third.

W.H.O emphasizes the need for a ban on tobacco promotion. (W.H.O. Expert Committee on Smoking Control 1978). It should be complete, fully implemented, well publicized, given major priority by governments and health authorities and continued on long term basis.(13)

Islam and Tobacco

Muslim Muftis and grand ulema have proscribed smoking tobacco soon after it was introduced to Turkey around 1000 H (1573 AD). Sultan Murad of the Othman Caliphate proscribed officially tobacco use and made it a capital offence in 1663. The previous grand Mufti of Saudi Arabia, Sheikh Mohammed bin Ibrahim cited in his fatwa the names of numerous grand ulemas and Muftis who proscribed tobacco use since its first appearance in Turkey and Maghrib (Morocco). From Turkey Sheikh Mohammed Alkhawajah and Sheikh Isa Asshahawi Al Hanafi; from Egypt Sheikh Ahmed Assanhoori Al Bohooti and Sheikh Ibrahim Allakani; from Maghrib Abu Al Ghaith. Al Gashash Al Maliki; from Syria Sheikh Al Najem Al Ghozi Al Aamri and from Yemen Sheikh Ibrahim bin Jamaan and Sayed Abubaker Al Ahdal. All the Saudi Arabia Muftis and Grand Ulema emphatically prohibited tobacco use in any of its forms, A tobacco smoker was lashed 40 lashes in Riyadh until quite recently. To mention but a few, Sheikh Mohammed bin Ibrahim and the present grand Mufti Sheikh Abdul Aziz bin Baz, al prohibited tobacco use, its promotion, sale, cultivation or dealing with it in any way other than destroying it.

The ulema and Muftis of Hadramout stood firmly against tobacco use. Imam Hussein bin Sheikh Abubaker bin Salim spent some of his wealth in buying tobacco farms and destroying the plant and replacing it with cereals, fruits and other crops. Imam Abdulla Al Hadad, Sayed Ahmed Al Hondowan, Sheikh Abdulla Basodan, Sayed Abdulla Asshateri and Grand Mufti Abdul Rahman Al Mashoor and many others considered tobacco use as "haram" i.e. prohibited.(56) Similarly, many ulemas of Mekkah and Medina e.g. Sheikh Abdulla Al Osami, Sheikh Mohammed bin Allan, Sayed Omer Al Basri, Sayed Saad Al Balky and Sayed Mohammed Al Barzangi, all considered tobacco use as haram.

The Ist International Islamic Conference of Ulemas on Drugs, Narcotics and Liquors held in Medina (Saudi Arabia) March 22-25, 1982, under the auspices of Crown Prince Abdulla bin Abdul Aziz passed a decree prohibiting the use of tobacco in any of its forms, its cultivation, manufacturing, trading, selling or promoting it in any way. It was considered as "haram" and the money gained through its production, selling, promotion or even taxation is "haram" and filth. Unfortunately, all Muslim governments paid no attention to these fatwas and continued in their previous policies. W.H.O., Eastern Mediterranean office published a book in 1988 under the title "Al Hokom Al Shari Fi Al Tadkhin" i.e Islamic jurisprudence decision on smoking" which involved the Fatwas of the ten leading ulemas of Egypt, explicitly considered tobacco use as "haram"(58)

All these Fatwas considered tobacco use as haram on the following bases:

1) It is detrimental to health. The Holy Quran states clearly "don't kill yourselves, (59) and in another aya", "and make not your own hands contribute to your destruction".(60) Islamic teachings and hundreds of Habiths (sayings) of the prophet (P.B.U.H.) encourage muslims to be in good health and abstain from things injurious to health.

   Some of the ulemas in the past were not sure that tobacco use was injurious to health and hence considered it as "Makrooh" i.e detestable, but not completely proscribed "haram". As more data was collected and the prestigious health organizations and authorities all over the world became more convinced with the deleterious and detrimental effects of tobacco use, all the ulema became convinced that it is "haram" and not only Makrooh. W.H.O. claims that tobacco use kills 2.5 million annually i.e ten times the number killed by atomic bombs of Hiroshima and Nagasaki of the second world war, and ten times the number killed by the AIDs epidemic in the last ten years.(58)

2) Tobacco use wastes huge sums of money. Saudi Arabia spends annually more than one billion riyals on tobacco imports.(61) Unfortunate poor Muslim countries spend more money on smoking and other tobacco use than the money spent on health promotion or education. In Bangladesh, a smoking member of the family robs his family of one quarter of their needed calories and food. About 18,000 children die annually in the country of malnutrition, directly related to tobacco smoking.

   The Holy Quran deplores those who squander wealth and property, "but squander not your wealth senselessly. Squanderers are indeed the like of satans."(62)

3) Smoking is bad and impure "Khabath", and the Holy Quran declares that the prophet (P.B.U.H) forbids all that is bad and impure and allows all that is good and clean. "He forbids all that is bad and impure and allows all that is good and clean. (63) Smoking is bad and impure, as it causes environmental pollution and is untidy. The smoke of the tobacco is unpleasant and can cause serious injury even to those who do not

smoke. Injuring others is completely prohibited in Islam and is considered one of the worst sins the Muslim can commit.

There is much need to keep the Muslim communities well informed about these fatwas, and the Muslim governments should stand firmly against tobacco use.

If Islamic teachings against tobacco are adhered to, Muslim countries and populace will succeed in avoiding the perils and tragic losses of life and wealth caused by tobacco consumption.

For Correspondence:
P.O. Box 11639
Jeddah 21461
Saudi Arabia (K SA)

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Sd/-
Dr. G.M. Malik

Ultrasonography in Chest Medicine

G.Q. Khan*; Iftikhar Bashir**

JK Practitioner 1996; 3:3 185-187

Application of ultrasound in chest was first described in 1964 on a patient with pleural effusion.[1] The use of ultrasound got subsequently well established in chest opacities.[2,3] Ultrasound is a reliable, efficient and revealing, imaging method to evaluate various clinical problems in the chest. It has been effectively used to guide a variety of interventions procedures in the chest.[4,5] The ribs, spine and airfilled lung usually act as barriers to ultrasound visualization of some intrathoracic lesions, tumour, consolidation or atelectasis in the lung provide ample sonographic windows for evaluation.

1. Sonography can differentiate pleural from parenchymal lesions. It can detect diseased parenchyma hidden by pleural effusion and can detect septations and other pleural abnormalities which are not otherwise suspected by other imaging techniques.
2. Ultrasound can also clearly demarcate the diaphragm and differentiate subpulmonic from subphrenic abscess.
3. Ultrasound being portanble can be used at bedside of the critically till patients.

Pleural Space:

Pleural space can be examined by ultrasound using either a direct or an abdominal approach. A high frequency (5MHz) linear transducer applied directly to the chest (direct approach) provides a broad near field and allows excellent visualization of the pleural space.

Abdominal approach:

The lower areas of the pleural space can be examined by use of lower frequency (3.5 MHz) sector transducer directed superiorly from the abdomen. The liver and spleen provide sonographic windows to the thorax. Fluid in the pleural space may allow for examination deep into the thorax.

Pleural fluid:

Direct approach: The direct approach allows even small amounts of pleural fluid to be detected by using a high resolution linear assay transducer. Most pleural fluid is relatively anaechoic and is easily recognized as an area of echolucency separating the paretal and visceral pleura.

Abdominal approach:

Pleural effusions are commonly detected during routine ultrasonographic examinations of the abdomen. The signs of effusion are as under:

- Hypoechoic fluid above the diaphragm
- Visualization of the inside of the thorax through the fluid collection

Ultrasound may be used to evaluate pleural abnormalities seen on chest X-ray. Focal pleural lesion may be loculated pleural effusions or solid pleural tumors.[6] Pleural masses are usually well-circumscribed and hypoechoic or of mixed echogenicity. The presence of internal echoes that do not move with respiration strongly suggests that the pleural lesion is solid.

Pneumothorax:

Pneumothorax is not easily detected by ultrasound. However when both fluid and air are present in the pleural space, pneumothorax can be recognized.

Diagnostic thoracentesis:

Ultrasonographic guidence of diagnostic thoracentesis can be done whenever clinically guided thoracentesis is unsuccessful or difficult[7]. It is especially useful when interpretation of chest radiography is difficult, when it is difficult to differentiate between subpulmonic or subphrenic collection. After examination of the chest X-ray the patient is examined sonographically to identify to largest and most accessible pocket of pleural fluid. The location and depth of the fluid is determined, and vital structures such as heart and aorta are identified. A safe site for thoracentesis is chosen based on ultrasound examination. The actual puncture of the pleural surface can be performed blindly.

Lung Parenchyma:

Ultrasound evaluation of the lung parenchyma is inhibited by the air in the periphery of lung. However, when the air spaces are collapsed or filled with fluid, the ultrasound evaluation is possible and can help to determine the nature of the lesion.

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* Associate Professor
** Postgraduate student.
Department of Medicine
Govt. Medical College Srinagar

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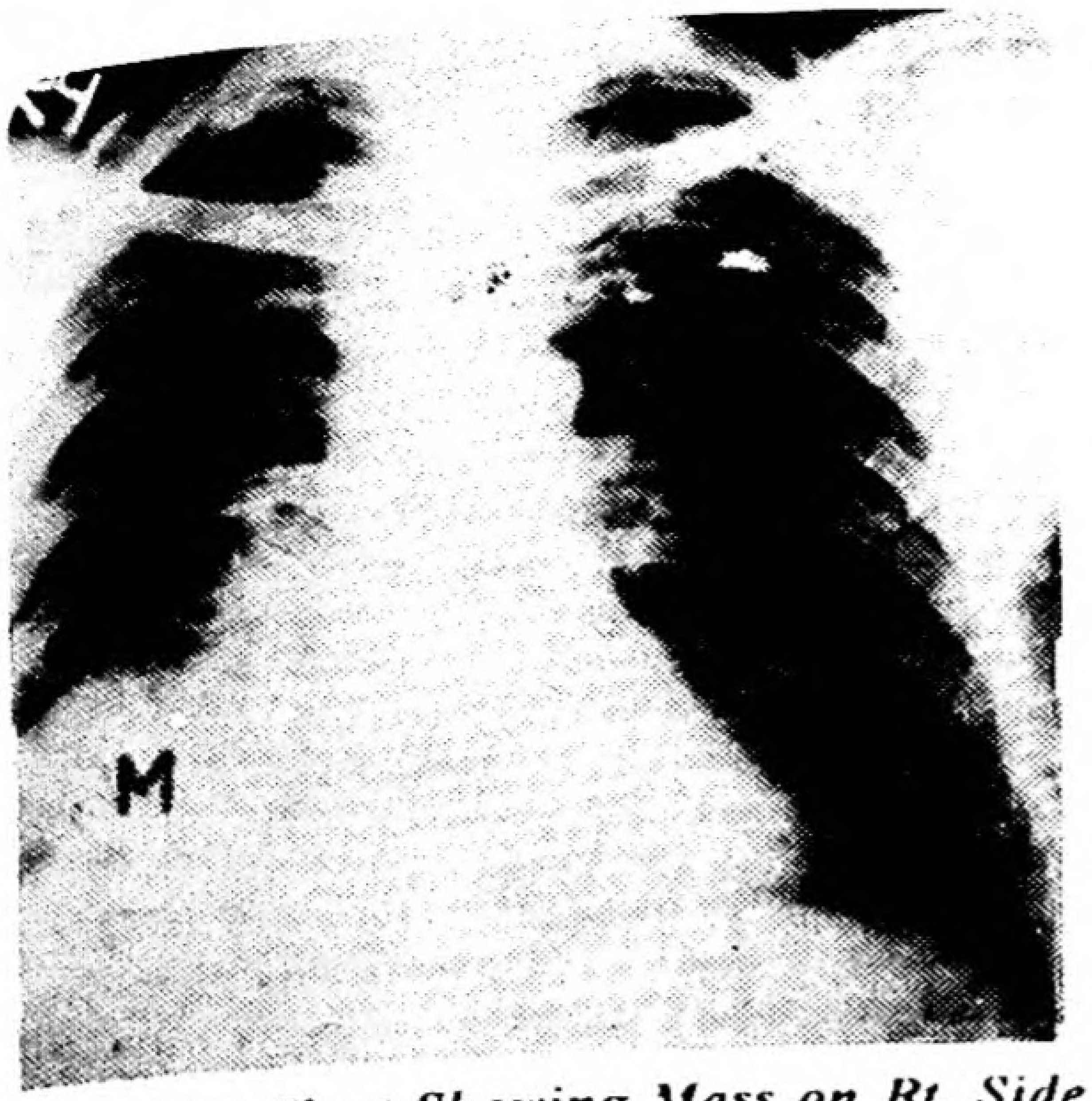

Fig. I - Xray Chest Showing Mass on Rt. Side

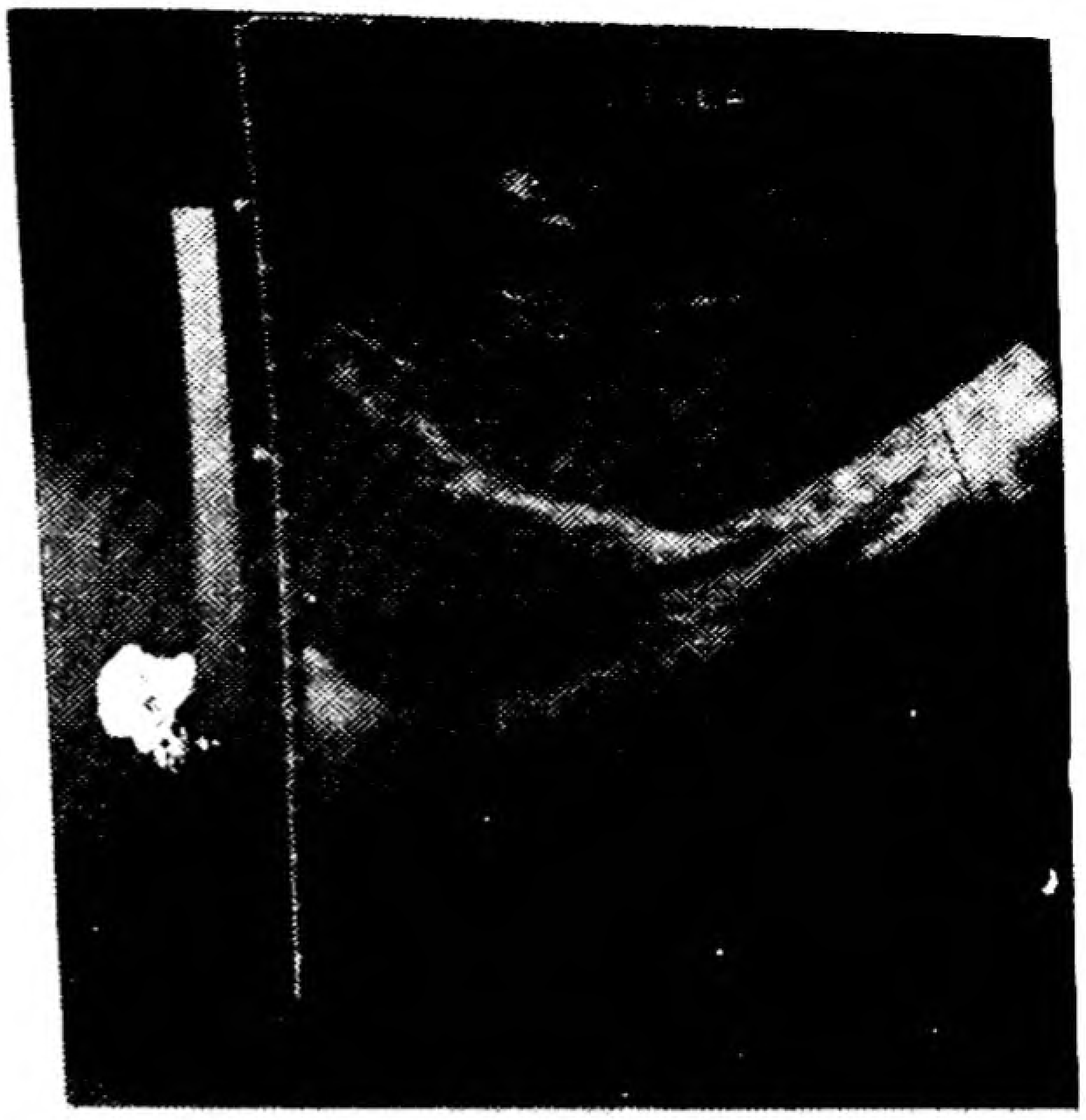

Fig. II - Ultrasound Showing Mass on Rt Side (Base)

Consolidation:

The air spaces of the lung are filled with fluid and inflammatory cells in consolidation and hence the airated lung is converted into a firm dense mass with good sound transmission.

Consolidated lung is hypoechoic as compared with highly reflective aerated lung and is usually hypoechoic as compared with liver and spleen because of its high fluid content. The consolidated lung is poorly defined and is wedge shaped.[8,9]

Air within the bronchi surrounded by consolidated lung produces highly reflective linear bronching echoes that can be recognized as sonographic air bronchogram[10].

Lung abscess:

A lung abscess is a localized suppurative process characterized by necrosis of lung tissue. Lung abscesses have thick irregular walls with echogenic debris and air within the internal fluid. With real time sonography a lung abscess will demonstrate expansion of its circumference with inspiration.

Lung tumor:

Lung tumors abuthing the pleural surface can be visualized sonographically as masses partially surrounded by aerated lung[8,9,11]. The margins of the tumor are often well defined (Fig. I & II).

Invasive procedures in lung:

Lung biopsy: Percutaneous transthoracic biopsy using flouroscopic guidence is a standard method of obtaining tissue diagnoses of pulmonary parenchymal masses[12]. However. flouroscopic visualization of lung nodules may be difficult or impossible when the nodules are located at the following sites.

1) Pleural base 2) on the ling apex 3) in the axilla 4) near the diaphrgm or 5) near mediastinum.

In such situation the ultrasonographic visualization of the masses is easy and best[11].

Ultrasound guided biopsy is quick. convenient. and safe. Because ultrasonography can visualize aerated lung adjacent to pleural based nodules better than fluoroscopy. the aerated lung can be avoided more easily[13]. The incidence of pneumothora for biopsy of pleural based nodules is 2% with ultrasound guidance as compared to 11% with flouroscopic guidance[11]. Both procedures have a reported 90% sensitivity for the diagnosis of lung cancer. Inspection of the chest X-ray is essential in planning the approach to ultrasound guided biopsy. Most lung lesions may be visualized and biopsied through the intercostal space. Lesions at the lung apex may be approached from either above or below the clavicle. Lesions on the diaphragm may be visualized and biopsied using an upward angle through the liver. Once the lesion is visualized a safe course for the needle is determined. The needle pass into the lesion may be performed either free hand or by use of a needle guide attached to the transducer. The patient is asked to suspend respiration while the needle is being advanced into the lesion. The patient may then resume shallow respiration while the needle is allowed to swing freely. The patient is again asked to stop breathing while the biopsy is taken. The specimen are immediately sent for cytopathological examination

In a study the utility of the fine needle aspiration biopsy guided by real time ultrasonography was studied in 25 patients with peripheral lung lesions[14] and the lesions were visualized in 22(88%) of patients and aspiration biopsy was performed in 20. Aspiration was diagnostic in 17(85%) of the 20 patients studied including 12 patients with malignancy and 5 patients with benigh lesion[14].

Fine needle aspiration biopsy under ultrasonography guidance is a safe, economical method with high diagnostic yield in the evaluation of peripheral lung lesions.

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Magnetic Resonance Cholangiography - a threat to diagnostic ERCP?

Magnetic resonance (MR) imaging is a new and non-invasive method of imaging the biliary tree. The present study evaluated the accuracy of MR cholangiography for patients with suspected bile duct obstruction.

Over a 6-month period one hundred and twenty-six sets of MR cholangiopancreatograms were assessed by two radiologists who were unaware of the clinical parameters, the other imaging tests and the final diagnosis. MR correctly identified 72 out of 79 patients with bile duct obstruction, giving a sensitivity of 91%, specificity of 100% and overall accuracy of 94% Thirty-two patients had common duct stones. MR correctly diagnosed 26, giving a sensitivity of 81%, specificity of 98%. Common duct stones were missed in six patients, in two of whom the stone can be seen on the MR cholangiogram retrospectively. Fourteen patients had malignant obstruction. MR correctly diagnosed 12, with a sensitivity of 86%, specificity of 98% and accuracy of 97%.

With the advent of laparoscopic cholecystectomy we need an accurate way of identifying common duct stones preoperatively. The skills and equipment for laparoscopic common duct exploration are confined to specialist centers and most surgeons prefer to have common duct stones dealt with by ERCP and endoscopic sphincterotomy before laparoscopic cholecystectomy. Depending on the selection criteria used for preoperative ERCP, a certain proportion of patients who do not have stones will undergo unecessary ERCP. The real question is: Should MR be used instead of ERCP when clinical and ultrasound evidence of ductal stones are equivocal? In the present study, MR missed ductal stones in 6 out of 32 patients (19%). This degree of sensitivity does not permit common duct stones to be excluded with confidence. MR will improve with experience and technical refinments.

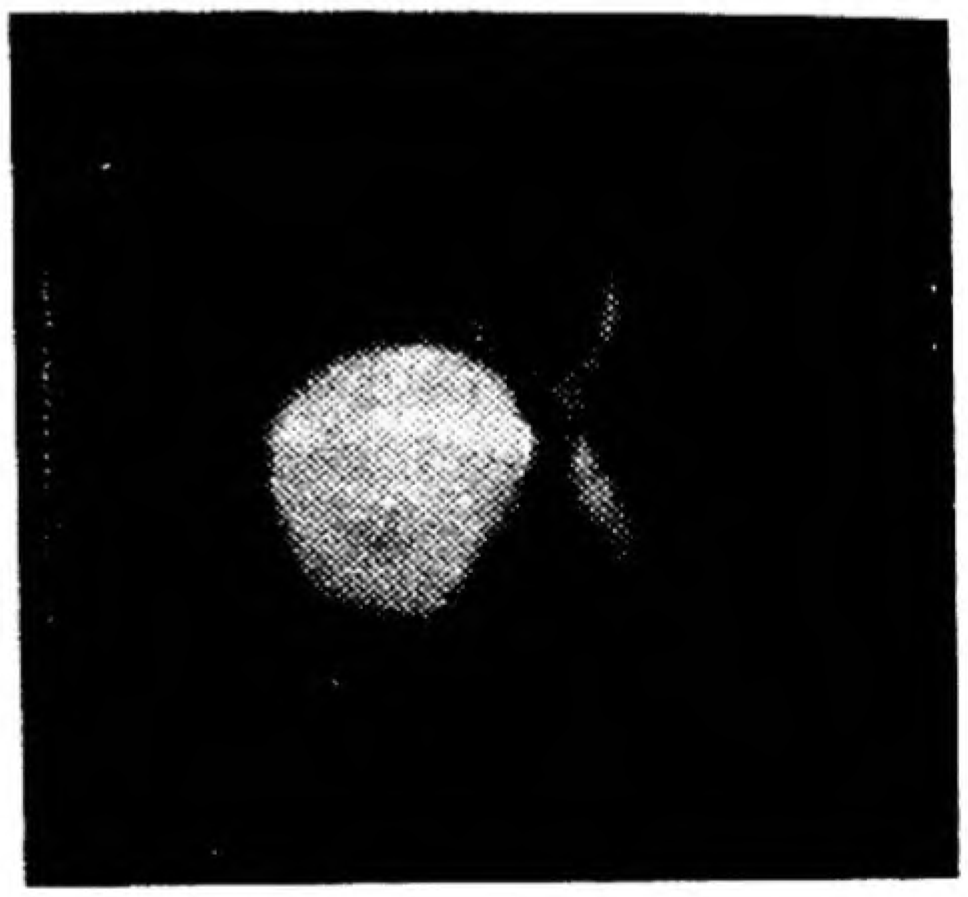

Figure MR cholangiogram: Can it replace ERCP?

When the accuracy of MR approaches that of ERCP, purely diagnostic ERCP will become obsolete and all ERCP procedures will be interventional.

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(Source: World Gastroenterology News 1/96, April)

Practitioner's Column

Laboratory diagnosis of HIV infection:

G.H. Hajini*; S.T. Hussain**

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Tests for HIV infection are used for three purposes: a) Screening b) diagnosis and c) surveillance, and these tests are directed towards the detection of HIV from body fluids and tissues, or detection of one of its components or antibodies to HIV from blood. Though the direct isolation of HIV remains the ultimate proof of infection but this is very difficult and expensive and is possible in only few selected and sophisticated laboratories. Antigen detection tests which detect cell free antigens in plasma or serum are less sensitive than culture and these antigens are mainly detectable in patients who have AIDS or are progressing towards ARC or AIDS. These are present over a short period around the time of seroconversion when antigenemia occurs. This too is rarely detected in African patients. About 30% patients of HIV infection have detectable levels of P24' antigens which increase to about 50% when samples are treated with weak acids which dissociate antigen antibody complexes. It has however been observed that subjects with detectable P24 antigens are 3 times more likely to show progression to AIDS than those in whom it can not be detected.

Antibody detection is the most satisfactory approach to the laboratory diagnosis of HIV infection. The tests are used to confirm clinical diagnosis, screen blood and organ donors and conduct epidemiologic studies. HIV antibodies generally appear within 4-8 weeks of infection. The standard screening tests have a sensitivity upto 99.5% with combined sensitivity and specificity upto 98%. Antimalarial antibodies do not seem to alter the test results. However these tests without confirmatory (supplemental) tests should not be used to identify asymptomatic individuals as being infected with HIV. Diagnosis can not always base on antibody detection tests as these are not present in very early infection- and may be lost in advanced AIDS. Patients with congenital infection and immune deficiencies may not seroconvert. Since these antibodies can also be passively acquired by neonates from seropositive mothers although only a small percentage of such neonates are truly infected, methods will have to be devised to distinguish true infection form the presence of antibodies from vaccination or passive immunotherapy.

It is fortunate that the spread of AIDS in early nineteen eighties was also by coincidence the period when advance in molecular biology and immunology provided theoretical basis needed for understanding the pathogenesis of the disease and the technical tools to detect the presence of HIV infection. The major technical advances which made this possible include a) production of monoclonal antibodies b) solid phase ELISA techniques for detection of antibodies directed against HIV gene products c) Western blotting d) polymerase chain reaction for detection of viral DNA/RNA.

Antibody detection tests:

Tests to identify antibodies against HIV became available in 1985 and were used initially for screening blood donors and this dramatically reduced the rate of transfusion induced HIV infection. Properly conducted enzyme immunoassay has about 99.8% specificity. False negative results are also known. False positive results do occur from intrinsic variations of such tests and has been estimated to be as high as 13% for antibody levels close to the cut off, so also can arise from heat treated, lipaemic and hemolysed sera; HLA antibodies against HLA, DQ3 and DR4 antigens; 19% of hemophilics; 13% of Alcoholics with hepatitis; 4% of hemodialysis patients; and 24% of patients with positive rapid reagin tests.

Tests for detection of HIV infection:

I) Antibody tests:

A) Enzyme immunoassay tests:
    a/ Whole virus lysate assay
    b/ Recombinant protein antigen
    c/ Chemically synthesized antigens
Other types of screening assays:
    a/ Particle agglutination assay
    b/ Haemagglutination assay
    c/ Latex agglutination assay
        d/ Solid phase immunoassays.
B) Supplemental tests :

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* Prof and Head, Deptt. of Dermatology
** Asst. Prof., Biochemistry
Medical College Srinagar
Kashmir - 190010, India

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a/ Immunoblotting (western blot)
b/ Radioimmunoprecipitation assay (RIPA)

II) Tests for viral antigens:

A) Enzyme immune assay (antigen capture)
B) Radioimmunoassay
  a/ antigen capture
  b/ competitive inhibition

III) Virus culture:

Tests to identify antibodies against HIV became available in 1985 and were used initially for screening blood donors thus dramatically reducing the rate of transfusion induced HIV infection. Not everybody needs HIV testing. Persons who should be offered such testing and counseling include.

a) Persons at risk for HIV infection like homo and bisexual men, I.V drug users, prostitutes, sex partners of infected persons or of persons at increased risk, haemophilics receiving clotting factors, and infants of mothers with risk of HIV infection.

b) Conditions associated with HIV infection. For evaluating patients with clinical or lab. evidence of HIV infection. Some infections which are found more often among persons infected with HIV as strep. pneumonae, H. influenzae, and mycobacterium tuberculosis.

c) Recipients of transfusion from 1978 to 1985 or till that time during which period no HIV testing of transfusion blood was carried out at different places.

d) Persons with other STD's. Out of 115989 samples collected from STD clinics 2.5% were reported HIV seropositive.

e) Patients of IV drug use; Such cases have high risk of HIV infection, 5% being positive in one survey.

f) Women of reproductive age: Women at risk include IV drug users, prostitutes, transfusion receivers, women with sexual partners infected with HIV, women living in communities with known high prevalence of HIV infection. Testing such women will reduce perinatal HIV transmission.

g) Hospital admissions: In high risk areas seroprevalence in admitted patients in one study tested 1.3% positive.

h) Persons with tuberculosis: All tuberculous infected patients should be HIV tested as this infection is frequently found in HIV infection. As high as 4% AIDS patients had tuberculosis while as the rate of HIV infection among persons with tuberculosis was 23% and 29% in two series.

Immune response to HIV-I infection:

People infected with HIV-I mount a vigorous antibody response to HIV-I and generate detectable antibodies within 3 months. prolonged seronegativity following infection is controversial. With advanced immunodeficiency antibody titre to most viral proteins diminish with the curious exception of antibody titres to transmembrane protein gp41. For many viral diseases antibodies induced by vaccination or as a result of primary infection protect against infection on subsequent infection but it is the cellular response that is primarily responsible for resolution of infection. HIV-I is unique in that the very cells that initials cellular response to HIV-I are the cells destroyed by HIV-I. HIV infects, functionally impairs and depletes a subpopulation of T-lymphocytes that express cell surface molecule CD4. This cell recognizes and induces functions in the immune response to foreign stimuli. Infection results in gradual CD4 T cell depletion, progressive immune unresponsiveness with effective paralysis of virtually all arms of immune system, increasing susceptibility to opportunistic infections and malignancies. The severity of clinical spectrum is reflected in a parallel severity of CD 4 T cell depletion.

Of various tests available for HIV diagnosis enzyme immunoassays (EIA) or enzyme linked immunosorbent assay (ELISA) for HIV antibody are the most widely used, and are a low cost, effective screening tests. Non reactive specimens are reported as negative. Reactive specimens are retested and then reported as repeated reactive but these are reported as positive only after confirmed by one of the supplemental tests. Since 1985 serodiagnosis and seroprevalence was started against HIV-I infection and after the discovery of first US native infected with HIV-2, HIV-2 serosurveys were started in 1989 in United States. In the beginning test centres relied on cross reactivity between HIV-I for HIV-2 antibodies to detect HIV-2 infected sample and it detected between 9% and 91% samples of HIV-2 infected samples. This necessiated the development of independent tests for HIV-2 antibodies. These kits became available in 1990. Subsequently kits were made available which could detect simultaneously HIV-I and HIV-2 antibodies in a single cell.

Several types of antigens are used in enzyme immuno-assays for HIV-I and HIV-2 antibodies and include whole virus lysate, recombinant proteins and chemically synthesized peptides. Whole virus lysate contains all the viral antigens as also the proteins of cells used to culture the virus, is therefore more sensitive and less specific compared to recombinant protein or chemically synthesized petide antigen which is less sensitive but more specific.

In this procedure antigen is bound to a solid support and then incubated with patients serum or plasma. Antibodies present bind to antigens which is detected with antihuman IgG which is labelled with alkaline phosphatase so that enzyme reaction will produce colour reaction proportional to the amount of antibody to HIV present.

Agglutination assays:

These are more widely used in Asia and Africa, are simpler and require practically no equipment, and are well suited for larger sero-surveys. Here diluted serum is mixed with a suspension of geletine particles coated with purified HIV antigen. Sera that agglutinate coated particles is labelled possitive. The test is however less sensitive and less specific. Also agglutination test, recombigen HIV latex agglutination assay, is costly, difficult to read and less sensitive.

Other rapid tests:

Several solid phase enzyme immuno assays where HIV antigens are immobolised on a membrane are referred to as rapid tests as the results are obtained in 10-15 min. Here antigen antibody immune complexes are trapped onto filters. Such complexes are then reacted with enzyme antihuman IgG conjugates and then a precipitating substrate. At the site of immune complex colour changes develop which stain the membrane, to be labelled as positive compared to control spots. Such tests are simple and rapid, but are costly. Here also false positive results are ruled out by confirmatory tests.

Supplemental tests:

As the initial serological assays yielded variable proportion of false positive tests, repeated positive reactions with same assays must always be confirmed by a supplemental test.

Western blot:

It is the most extensively used conventional supplemental test which uses multiple antigens of HIV. A positive test is defined as one which demonstrates antibodies to all the three major gene products of HIV (gag, pol, and envelop proteins) and is a conclusive evidence of HIV infection. Any pattern which dose not fall in positive or a negative category of western blot is labelled as an indeterminate pattern, which could be due to the antibodies that cross react with one of the proteins of HIV or that the individual is infected with HIV and is in the process of mouting a classic antibody response. Such a test is repeated after one month to see if the indeterminate pattern is a pattern in evolution. Thus a patient with a positive ELISA but negative western blot is a false positive ELISA reactive. But if the indeterminate pattern continues for six months in absence of epidemiological risk factors for clinical findings it may be considered negative for antibodies to HIV. However confirmation in such cases comes from polymerase chain reaction (PCR) which is of two types a) DNA, PCR b) RNA, PCR.

WB assays use partially purified HIV proteins obtained from viral lysates immobolised on nitrocellulase paper to determine which antibodies in the sample are responsible for the reactivity observed in ELISA. Viral proteins are fractionated by gel electrophoresis. A sheet of nitrocellulase is applied to the gel surface, and with electric current the proteins move from gel to the nitrocellulase sheet, which is then cut into strips. Repeated reactive serum or plasma is then incubated-with strip. Human antibodies to HIV proteins if present will bind to the antigens. Such strips is then incubated with goat antibody against human IgG that has been conjugated with an enzyme say alkaline phosphatase. The conjugate will bind the immune complex. Appropriate substrate is then added. Coloured bands appear if human IgG was present on strip. It is compared to control. It is seen that antibodies to gag protein appear first on W.B. test, and these antibodies decrease or become undetectable with the onset of clinical symptoms. But antibodies against glycoproteins and transmembrane proteins can be detected in nearly all HIV-I infected patients regardless of clinical stage. False positive rate for combined ELISA and WB is reported less than 1:100,000.

Immuno-fluorescence assay:

IFA is a low cost alternative to WB as a supplemental test. HIV infected lymphocytes are inactivated and fixed on a slide. Uninfected cells serve as a control. Diluted serum is incubated with cells and then washed & incubated with fluorescein isothiocyanate laballed antihuman IgG. Positive and negative control sera are used side by side. Antibodies in positive sera bind viral antigens. Test is reported on the basis of percentage of fluorescent cells and the pattern of fluorescence. Studies have shown good concordance between IFA and western blot results.

Radioimmunoprecipitation assay:

RIPA is also some times performed as a supplemental test. Cells infected with HIV-I or II are exposed to radioactive amino acids that become incorporated into the viral proteins. Cell lysates from these cells are first cleared of proteins which bind nonspecific antibodies. Then the lysate is mixed with patients serum and incubated. The immune complexes are then absorbed with protein A sepharose beads. Radioactive antigen antibody complexes are eluted and positive elutes are separated by gel electrophoresis to determine specific viral proteins. The binding patterns are similar to those of western blot. The procedure is cumbersome and expensive though it is more sensitive than WB for antibodies to Env.glycoproteins (gp60, gp120). RIPA is very helpful in confirming HIV infection and in typing the infecting virus as HIV-I or HIV-II

Assaying HIV antibodies in dried blood specimens:

It has recently been shown that HIV antibodies can be detected in blood samples of newborn as IgG antibodies are transfered to foetus. Thus blood samples from newborn can be used to study prevalence of HIV in women bearing live born infants. Testing dry blood samples collected on filter paper from finger sticks give the same sensitivity and specificity as testing serum and plasma provided recommended precautions are properly observed. Repeatedly reactive specimens are confirmed by testing with western blot.

Tests for HIV-IgA and IgM antibodies:

Detection of HIV infection in infants born of sero positive mothers by serological tests is difficult as HIV reactive antibodies produced by infants are obscured by large quantities of maternal IgG. These passive antibodies may persist for about 18 months in infant. Thus those tests that can differentiate maternal from foetal origin have a diagnostic value. Hence assays specific for IgM and A are essential as these antibodies do not cross placenta./ This is accomplised by an immunoblot test after removal of IgG by repeated absorption of IgG and using class specific (for IgA & M) antibodies for preparing conjugates. IgA antibodies have been detected in 66% of infected infants at the age of 6 months compared to only 33% of IgM antibodies at the same age.

HIV antigen detection:

In 1986 enzyme immunoassay tests for HIV core antigen (p24) became available for detecting viral antigen in serum, plasma and CSF. In this test the patients specimen is incubated with anti HIV-I capture antibody bound to a solid support such as polystyrene beads of microplate wells. After washing the immune complex bound to solid support. further incubation is done with a second anti HIV-I antibody to form a sandwich. This second antibody in some assays is conjugated to an enzyme that subsequently is reacted with substrate. The resultant colour change can be detected by increase in absorbance at a specific wave length which provides a quantitative measurement of the amount of HIV antigen present in the test sample. Other assays use unlabelled second antibody (Rabbit anti HIV-I) followed by enzyme conjugated goat anti rabbit IgG. After the final wash the amount of HIV-I present in immune complex is detected as before by colour change resulting from enzyme conversion of substrate. This type of assay is more sensitive.

All reactive specimens are confirmed by specific neutralization assay to verify the presence of HIV-I antigen.

HIV antigen has been detected in early infection of seronegative individuals who are members of high risk group or who present with acute HIV syndrome before seroconversion. However most HIV infected individuals became HIV antigen negative shortly after seroconversion.

Antigen testing is useful in a) corelating the onset of antigenemia and the progression of disease b) determining HIV infection in neonates born of HIV infected mothers c) as an effective tool for assessing the therapeutic efficacy of different antiviral drugs.

It has been shown that the sensitivity of test increases with clinical progression of disease showing 4% positively in asymptomatic patients, 56% of patients with ARC and 70% in AIDS patients.

Detection of HIV infection by PCR:

Because of the low frequency of HIV infected peripheral blood mononuclear cells (PBMC)in a seropositive person, conventional techniques not being sensitive enough could not detect HIV proviral DNA directly form patients lymphocytes. To detect it, DNA must first be amplified to detectable levels. This was achieved by using polymerase chain reaction. Here PBM cells are incubated with nonionic detergents and finally treated with proteinase K. After heat treatment the resulting DNA preparation is ready for PCR usually 1 ug of DNA (Equivalent to about 150000 mononuclear cells) is used per PCR reaction. DNA has a double helix in which each strand can serve as a template for the synthesis of other strands and the two strands can be separated by simply heating DNA. The process is repeated to synthesize larger number of daughter DNA molecules which can then be easily detected. RNA can also be amplified by using reverse transcriptase to produce DNA complementary to RNA and then amplifying the DNA.

The process is so sensitive that special precautions have to be taken to prevent DNA molecules floating in air or present at pipette tips from contaminating the mixture and getting amplified, thus giving false results.

PCR has demonstrated both clinical and research utility for:

1- Direct detection of HIV DNA or RNA from cells of infected persons.
2- Detecting infected persons during the window period (i.e before the generation of HIV specific antibodies)
3- Resolving the infection status of individuals with an indeterminate W.B.
4- Screening of neonates for HIV infection
5- Distinguishing HIV-I from HIV-II infection
6- Defining the patterns of transmission & evolution of virus throughout the population.

Virus culture:

Serosurveys are capable of identifying prior exposure to HIV, but can not demonstrate if the person is currently harbouring the virus. Although virus culture is a poor diagnostic tool because of its relative insensitivity, high cost, lengthy time requirements, but it is a standard method for judging other diagnositc tests and identifying variants. Reported rate of virus isolation from blood of HIV-I antibody positive persons range form 65% to 100% and has been occasionally isolated from lymphoid issues, plasma, saliva, semen, tears, brain tissue, CSF, breast milk and urine, however there is no evidence that contract with saliva, tears and urine has resulted in infection. High isolation rates of virus from PBMCS promote the use of whole blood as a principal source of diagnostic material. Negative cultures are maintained and monitored for 4 weeks.

Haemotological and Immunological tests:

While the serological tests are most important for screening, diagnosis and surveillance of HIV infections many haematological abnormalities have been found in AIDS patients. A high ESR is present in all patients with AIDS but this is a non-specific abnormality. More useful for staging and evaluating patients with AIDS are complete blood counts (including differential WBC counts), platelet count and T Helper (CD4+)/ T suppressor (CD 8+) cell counts. WHO has proposed a system where patients after clinical staging (clinical axis) are further put into three subclasses based "on laboratory axis"

Subclass A" Total lymphocyte count 2000/m^3 or if CD4+ counts are available then CD4 + lymphocyte count 500/mm^3.

Subclass B: Total lymphocyte count 1000-2000/mm^3 or CD4 + lymphocyte count 200-500/mm^3.

Subclass C: Total lymphocyte count <1000 or CD4+ lymphocyte count <200/mm^3.

For surveillance lympocyte and CD4 cell counts are used to provide evidence for AIDS in a child <15 MO of age whose mothers had HIV infection during perinatal period and whose serum is positive for antibody. In such patients reduced total lymphocyte count, depressed CD 4 (T helper) lymphocyte count and decreased CD4/CD8 ratio are considered to support a diagnosis of HIV infection as long as subsequent antibody tests (Western blot, immunofluorescence) if done are positive.

Flow cytometry for counting the number of different types of lymphocytes (Immunophenotyping) has been very useful in monitoring extent of Immunological damage and predicting survival in AIDS patients.

The series of articles on AIDS is not a comprehensive review but basically intended to guide both under and postgraduate students of Medicine. For further reading following references are suggested.

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"The Psychology of Resuscitation"

Ralph M. Shenefelt

JK Practitioner 1996; 3:3 193-194

Sixteen years of teaching and performing resuscitation has provided me a useful logic system that can be utilized when teaching or performing resuscitation. It has been useful to others. perhaps it will be useful to you.

I. Lay people and some professional experience fear and anxiety in regard to resuscitation attempts.

A. This is often the result of a mental thought process which goes something like.
  1. If I do the wrong thing
  2. The patient will be made worse
  3. If they are made worse as a result of my actions.
  4. They will die
  5. If they die as a result of my actions, then I will get sued
  6. If get sued. I will lose money

II. Well meaning (but misguided) instructors and rescuers often focus on errors

A. Doing that will break ribs and cause internal injuries

B "Doing that will make the patient worse."

C " That is a dangerous mistake."

III Communication of this type causes learners and rescuers to:

A. Fear doing something wrong. thus reinforcing anxiety.

B. The result:
  1. Learns become timid in resuscitation attempts which in turn
  2. Reduces effectiveness of technique
  3. Decrease blood flow and oxygen delivery
  4. Decreases likelihood of resuscitation attempts or resuscitation success

IV. Death is the greatest degree of poor health.

A. The pulseless patient is essentially dead.

B. No further harm can be done attempting to resuscitate one who seems deceased.

C. You cannot make some one who is dead, more dead.

V. Communicating properly helps.

A. CPR should not be given when a pulse is present.

B. CPR does not keep the heart beating.

C. CPR artificially circulates blood and oxygen when the heart is stopped

D. CPR buys time until a defibrillator or definitive care is available.

VI Consider the psychological impact of terminology.

A. "I did CPR on a patient and they died.

B. "I did CPR on a patient and they stayed dead."

C. Statement "A" implies a fault of the rescuer, while statement "B" implies no change in patients's condition from the resuscitation attempt.

D. Rescuers should not hold themselves responsible for a resuscitation attempt that is not successful. The vast majority are not.

VII. Using correct terms when communicating about resuscitation attempts provides:

A. A more realistic appreciation for resuscitation efforts;

B. Less concern about injuring an arrest patient;

C. A more positive and low stress learning and resuscitation environment

VIII Rescuers who fear injuring a patient with CPR are less likely to perform well or perform at all.

A. As a result, care may be less than adequate.

B. Delays in providing CPR or inadequate effort significantly reduce the possibility of successful resuscitation and greatly increases the likelihood of severe brain damage

C. A rescuer who has a realistic understanding of resuscitation
  1. Is more likely to provide rigorous resuscitation and thus
  2. Increase the patient's chance for a meaningful recovery

IX. Therefore. when teaching or communicating about resuscitation:

A. Be aware of the psychological effects of your words.
  1. Avoid phrases that cause fear and anxiety such as;

    a. "Doing that will make the patient worse."
    b. "If you do/did that you kill/killed the patient "(This is a particularly destructive statement which strikes at an emotional rather than intellectual level. It is a malicious and unnecessary distortion)
    c. That is a dangerous mistake."
2. Instead, use positive comments, such as:
    a. " This technique will give the patient the best chance of being revived."
    b. "Mistakes do not `kill' the patient, but they might cause complications for other rescuers. Let's try that again... this is the correct

X. Use positive reinforcement.
A. Emphasize what went right.
B Remember that "flawless" CPR is unnecessary.
C. Focus on preserving the brain & reversing ventricular fibrillation.
D. It takes a caring spirit to keep trying. Caring takes courage. Caring requires a loving heart.
E. "Death is nature's advice to get plenty of life." (Gothe)

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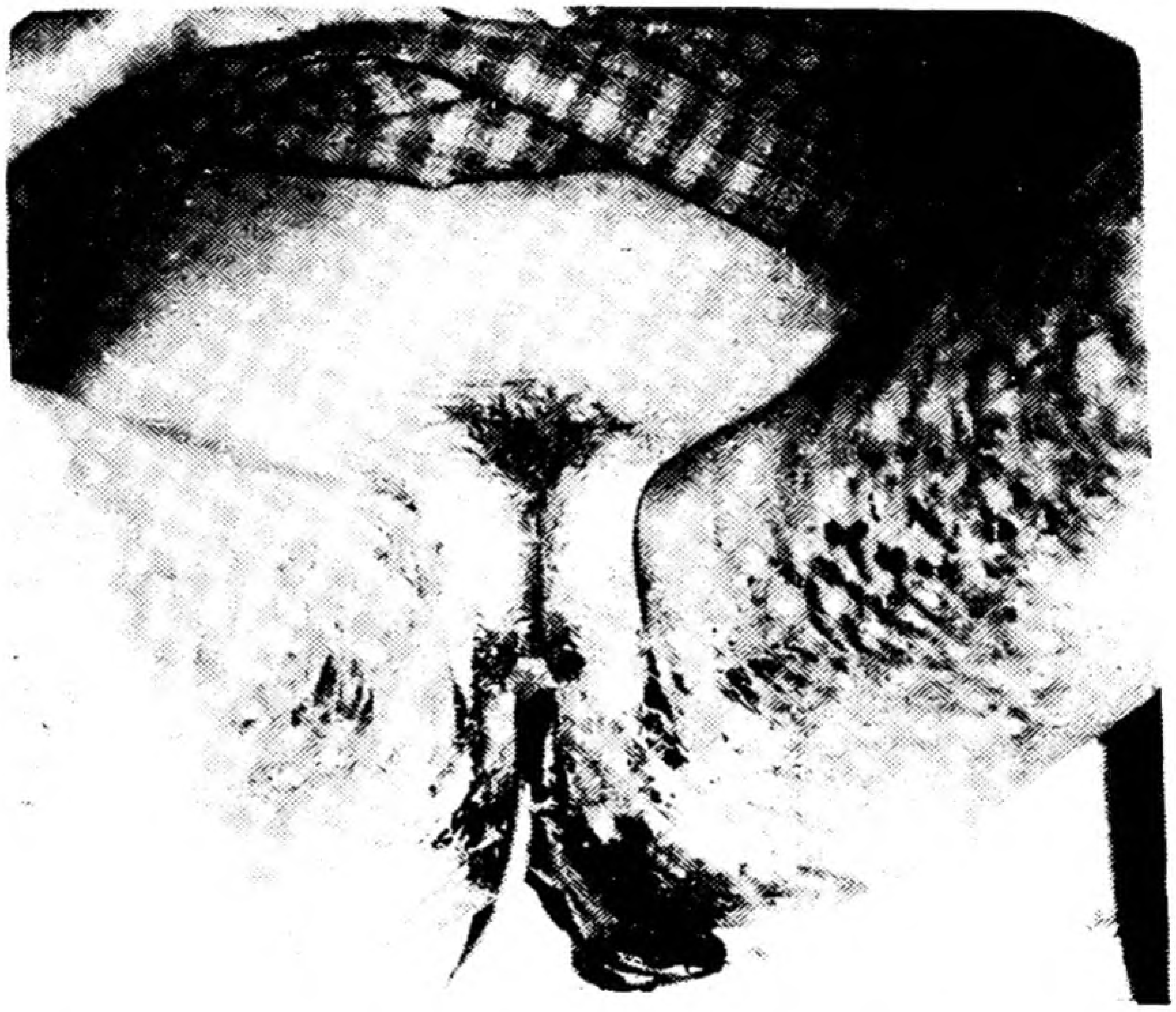
Clinical Images



"A Rare Complication of D&C"

JK Practitioner 1996; 3:3 194

Mrs 'Z', 35 years old Muslim lady reported to the emergency surgical services of SMHS Hospital, Srinagar kashmir (India) with history of passage of faecal matter through vagina following some surgical intervention for termination of 3 months pregnancy performed by an unqualified "daya" at her residence. On examination gangrenous gut with multiple perforations discharging faecal matter could be seen. After an emergency laporotoray patient had a huge perforation of the fundus of uterus through which the gangrenous gut having multiple lacerations could be easily retreated back for which a resection with end to end anastomosis with closure of uterine perforation had to be performed. This rare complication after so called "D&C" for termination of pregnancy is very rarely seen nowadays.

Author:
Mir Nazir Ahmad
Asst. Professor Surgery
Medical College Srinagar

Implementing and Sustaining Health Service Quality

Syed Amin Tabish

ABSTRACT: Total organisation commitment to continuously improve the quality of the patient care is the central concern of health care institutions. This commitment is woven throughout the fabric of the organisation. TQM aims at developing humane work systems and investing in people. It is critical to begin with a firm understanding of users of the service. The design be developed into an operational plan alone with processes necessary to create the service. Patient statisfaction and needs be constantly monitored. An ongoing. comprehensive self assessment system, supports and promotes continuous improvement in the quality of patient care. It is particularly important that quality care be ingrained as a philosophy in the organisations to achieve excellence in health care.

Key Words: Quality Management, Excellence, health care, organisation, Accountability, Evaluation.

JK Practitioner 1996; 3:3 195-197

INTRODUCTION

Quality management has become a major concern in the delivery of health care. The demand of the community, economics of medical practice, increasing legal action in malpractice cases and concern to protect the interest of patients has focused the attention on quality management programmes for all hospital administrators. Pressures to apply principles of Total Quality Management (TQM) to health care organisations is ever-increasing when the health care industry struggles with demands for accountability. New and distinct measuring and reporting methods are being designed while preserving the uniqueness of health care profession.[1]

TQM aims at developing humane work systems and investing in people. It combines good team work and motivation with well developed skills in statistical analysis and detecting variations in practice over and above normal limits.

ORGANISATIONAL COMMITMENT

The hospital has a primary obligation and both a legal and moral responsibility to see that the quality of care meets acceptable standards and that the interests of the patients are paramount above all matters.

Quality of health care services means considerably more than technical excelllence. It means providing the community with a product that really serves the health needs of the individuals and the community at large. It also means conservation of scarce community resources while meeting those needs. High quality service starts with high managerial ethical standards.

The organisational commitment to continuously improve the quality of patient care is the central concern of health care organisations. This commitment is woven throughtout the fabric of the organisation, appearing in strategic planning, allocation of resources, role expectations, performance evaluation, reward structures, and the role of the organisation in the community.

ELEMENTS OF QUALITY MANAGEMENT

It is important to conceptualise and impletment quality management in terms of systems:

there must be a system for establishing professional standards-standards of behaviour, standards of organisation, standards for measuring professional practice, etc.

there must be a system of surveillance so that one can know if the predetermined standards are being met

there must be a system of enforcement of those standards.

THE DIMENSIONS OF QUALITY EFFORT

Some dimensions have been identified to pursue in the early phase of implementing quality leadership[2] :

the education, re-education, and active leadership of top management

a two year strategy for starting and implement-

MAMS FIMSA FRIPH
Department of Public Health Medicine
University of Bristol
Bristol, (England)
BS8 2PR, UK.

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ing a quality improvement philosophy
a network of co-ordination, guidance, and technical support
an organisational culture supportive of quality leadership
training and education for all, and
carefully selected improvement projects.

WORKING TOGETHER

Quality patient care is provided by a complex blend of multidisciplinary technological and human resources. Additionally all activities must operate in rapidly changing environment that imposes a broad range of pressures on the provider of services, as well as the recipient.

Quality improvement is hard work and takes a long time. Major gains in quality and productivity most often result from teams-a group of people pooling their skills, talent and knowledge. With proper training, teams can often take complex and chronic problems and come up with effective, permanent solutions. Another advantage is the mutual support that arises between team workers. Working together promotes accountability.

It is important for the teams to understand to view tasks as steps in some process instead of unrelated events. In doing so, they will come to understand why quality can be defined only by the people who receive the out-put (service or product) of the process. They must learn where to look for problems in the process, and which problems they have the capability to change.[2]

SCIENTIFIC APPROACH

The core of quality improvement methods is scientific approach (systematic way for individuals and teams to learn about processes, agreeing to make decisions based on data, to look for root causes of problems, to seek permanent solutions). The tools of the scientific approach include[2] :

flow charts
pareto charts
cause-and-effect diagram
operational definitions
stratification and is/is not analysis
time plot
control chart
dot plots and stem & leaf displays
check sheet
scatter diagram.

Evidence suggests that staff can develop their own guidelines locally, and despite deviation from the "ideal" of a national guideline patient outcomes and staff performances can still be seen to have improved.[3]

This suggests that factors other than acquisition of the most recent scientific knowledge affect staff behaviour and patient outcomes. Certain key aspects of knowledge must be identified before local groups can be effective.[3]

ACHIEVING EXCELLENCE

To achieve excellence in a health care system patients, providers and systems have to be involved in employing the principles of TQM.[4]

Patients Involvement - in preservation; participating in quality care process through education and utilisation; in evaluation of functional status before, during and after intervention; in satisfaction; in clinical outcome.

Provider Involvement - in planning, implementing, analysing and educating; in defining guidelines; in reassessing and continually modifying the care map, always striving for quality care.

System Involvement - in providing structure and mechanisms, support staff, and information systems and being willing to focus on quality as a part of its mission.

SUCCESSFUL CHARACTERISTICS

The traits of America's most successful companies as determined by Peters[5] are :

closeness to customer
productivity through people
autonomy and entrepreneurship
constant innovation
belief in team work

EVALUATION

The attributes of quality which can be used for assessment include :

access
continuity
comprehensiveness
effectiveness (for individual patients, at provider level, at population level)

At the core of the quality must be the effectiveness and efficiency of interventions which should become a routine part of assessment and activity.[6]

FUTURE ISSUES

The challenge for the future is :

- to increase the amount of health technology assessment carried out, and
- to develop methods of ensuring that health

care converges with the best practice - the promotion of Evidence Based Medicine.[7]

CONCLUSION

To achieve excellence TQM should become a continuous part of planning process of health care institutions driven by the goal of satisfying the needs of the patient. Management at all levels must constantly study the process it controls, seek ways to improve it, organise an appropriate team to address the issues, determine what data are necessary, collect the data, test the data, implement the change, and evaluate what occurred.

Quality improvement is a philosophy based in attitudes that govern the patterns of thought and behaviour. TQM needs total quality mindset. Real effectiveness will come only when it is coupled with creating attitudes of contribution, team work, leadership, unfailing human interest and courtesy.

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Young Scientist Award in Medicine 1995

Syed Mohd Saleem Kamili, a PHD students in the Department of Gastroenterology SK institute of Medical Sciences was awarded the State Young Scientist Award 1995 by Department of Science and Techonology, Govt. Of Jammu & Kashmir for his outstanding and original research work on hepatitis E. The award was presented by the Advisor to Governor, Mr. R.K. Sayeed in an impressive ceremony at Jammu on 19th April 1996. Mr. Kamili has recently submitted his PHD Thesis entitled "Hepatitis E: Studies on transmission, etiological agent and seroepidemiology" carried out under the supervision of Prof. M.S. Khuroo, to the University of Kashmir for the award of PhD degree in Biochemistry.

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Future Prospects for International Medical Graduates in the United States

Tanveer P. Mir*, M.D., F.A.C.P.;
Farooque A. Khan**, M.B., M.A.C.P.

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The graduate medical education reform in the United States has had a profound impact on the availability of residency position to International Medical Graduates (I.M.G.). Historically, medical education in the U.S over the past few years has led to a large number of unemployed physicians from different parts of the world. The National Residency Matching Program (N.R.M.P) has shown a constant change over the past few years (Table I). 1988 was labeled as the 'Black Day' in the history of NRMP in the U.S. Several programs in the primary care programs of Internal Medicine, pediatrics, family practice had unmatched positions. The situation in New York and New Jersey looked particularly grim. What followed in the subsequent years was a decline in interest of the U.S. medical graduates in these specialties.

Since this opened up hundreds of vacancies, the immediate answer was to resort to the available pool of IMGs to fill the slots. This trend continued over the next few years till 1994, when the Graduate Medical Education (G.M.E) reform and the Generalist focus came into being. At this time the Council on Graduate Medical Association (C.O.G.M.E), and the Association of American medical Colleges and other societies presented the impact of overspecialization on the healthcare industry and suggested an emphasized approach to primary-care programs. This included several incentive programs like the loan-forgiveness program for primary-care specialties for the USMGs and H-I visa opportunities for IMGs seeking training and J-I waivars for the IMGs seeking a job in the primary-care areas. Several outreach programs were initiated and hospitals initiating primary care programs received financial incentives in the form of grants or 'UPWEIGHTEE' status. This meant that these programs received a higher reimbursement for every resident trained in primary care. This led to and influx of USMGs in the primary care specialties as reflected in the change in matching trends in the primary-care specialties (Table II).

The most dramatic change in the country was noticed in the family practice area, where 78% of the total 3137 positions were filled by USMs. The changing trend from 1992 to 1996 can be appreciated by glancing at Table 3. Suffice it to say that the trends in New York City do not match the national numbers, but the trend of USMs filling primary-care slots is on the rise. The grass is not green for IMGs anymore. The competition is fierce, and even the IMGs who match in programs either make it because of extremely high scores or research background.

For a second year in a row, more than 50% of the nation's 14,539 Medical school seniors chose to pursue a training in either Internal Medicine, family practice or pediatrics, according to the results of the 1996 match. Family practice showed the most dramatic increase of 9.1% increase over the past year. The most startling drop occurred in Anesthesiology, which experienced a 53.8% drop from last year. Diagnostic Radiology also experienced a decline of 26.6% and Pathology a drop of 14.9% as compared to last year. According to Dr. Jordan Cohen, president of the American Association of Medical Colleges, the growth in Internal Medicine, combined with anecdotal evidence that subspecialization is on the decline, demonstrate that the specialty is moving toward primary care (1). Because of an oversupply of physicians as addressed all experts (2,3,4,5), residency positions may be capped soon, and funding for IMG residents could be targeted (1). Data from the AMA and American Osteopath Association indicate the disproportional increase in physicians to the changes in population (6,7) IMGs, who in the past few years have added

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*Association Program Director,
Department of Medicine
Chief, Division of Geriatrics
Nassau Country Medical Center
Assistant professor of Medicine
State University of New York at Stony Brook
**Chairman, Department of Medicine
Nassau Country Medical Center
Programs Director, Internal Medicine
professor of Medicine
State University of New York at Stony Brook

significantly to the growing pool of physicians in this country, seem to follow the same path as USMGs have chosen subspecialty training over primary care (8,9,10.)

Revamping medical education seems to be an integral part of the health-care reform. Some of the changes included in the reform are downsizing in training programs, drastically reducing subspecialty fellowship positions particularly in Cardiology, Gastroenterology and Pulmonary Medicine and last but not least curtailing the number IMGs in training . Changing market forces have led to reduction in specialist needs in the future. The pool of IMGs competing for a diminishing pool of residency positions is increasing and these large numbers cannot be absorbed into the existing positions in 1996.

NCMC EXPERIENCE:

The 1996 recruitment year is a memorable one in the history of the Internal Medicine recruitment program at NCMC. NCMC is 615-bed tertiary care hospital on Long Island, N.Y. The program in IM is comprised of 70 residents and 30 subspecialty fellows. The 30 first-year positions are offered through the match. This year the department received over 6000 applications for these positions. After careful screening about 500 candidates were interviwed, and 30 accepted through the match. The ratio of number of applicants for each position was over 200. These numbers do not reflect the telephonic requests, post-match applications, and other candidates showing up on a daily basis, seeking a position. We have monitored the recruitment trends over the past 10 years or so and this year seems to stand out in comparison.

CHANGE IN IMG MARKET:

Based on the number of candidates who continue to take USMLE, these numbers will grow to astronomical proportions each year. Each day brings in a fresh pool of IMGs seeking positions in IM or other fields. Whatever the incentive-better training facilities, job opportunities and financial security-U.S. training had to offer ,the scenario has changed for the worse for new IMGs in the US. The grass isn't green anymore. As mentioned above, this is a simple supply and demand situation. At a time like this when the ivy towers of U.S. medical schools are no longer insulated from the health-care marketplace, an acceptable solution to the problem would be to curtail the number of IMGs that are being trained. The 125 medical schools in the US graduate about 16,000 students each year. The total number of first-year positions is 25,000. The IMGs fill the 7000 positions each year and it is felt that they continue to stay in the U.S. either as subspecialty fellows or add-on to the physician oversupply. One of the suggestions is to close some U.S. medical schools as a way to curtail the supply doctors. This is not well accepted by medical school faculties and executives. "All sources of Government funding for graduate medical education should be allocated in a manner consistent with the counsel we are giving to American medical schools. Policy-makers may accomplish this and ensure the continued excellence of the physician work force by limited government funding for residency training to graduates of accredited U.S medical schools"(II)

E.C.F.M.G.

The ECFMG has been offering the 'entrance examination' for residency training in the U.S. since 1958. Traditionally, its role has been one of providing a standardized examination to IMGs to evaluate their credentials. After going through different forms of examinations and techniques, it currently offers the USMLE parts I (Basic sciences)., II (Clinical component), and III (Licensure eligibility). Presently parts I & II are required to enter residency training in the US. In the near future an additional clinical competency and linguistic skills component consisting of ten different areas of knowledge, clinical skills and patient evaluation and management is being introduced. The cost of developing this format and offering it at centers restricted to the United States will make it extremely difficult, rather impossible for IMGs to come to the U.S. for training. As of 1997, this will be a uniform requirement for all candidates applying to residency training positions. The visa constraints and an approximate $2000 examination fee eliminate most candidate. The ones who will make it through this marathon may yet not have the assurance of a residency position, since the market-forces may be different. It is vital for candidates to known that maintaining the balance on this tight-rope to the end may not bear fruitful results.

CONCLUSIONS:

1. The changing market-forces and introduction of managed-care has led to a reduced need for specialists in the USA.
2. Increasing numbers of IMGs are competing for a total of positions left unfilled by the USMGs in the 1996 match, and this trend will continue to worsen in the future.
3. Those IMGs who have no visa restrictions and do not require extra programmatical support

have added advantage . Several programs do not entertain non-immigrant IMG applications.

4. Based on Institute of Medicine and Council of Graduate Medical Education predictions, the future for IMGs in the U.S. appears to be bleak.

ADVICE:

a) Obtaining an ECFMG certificate does not guarantee a job or training position in the U.S.

b) Specialty training positions like Cardiology/ G.I/ Pulmonary Medicine are being drastically reduced and opportunities will be minimal if no non-existent .

c) Obtaining residency position without a personal interview with the faculty in the U.S. will be virtually non-existent in the near future.

d) The few IMGs who will secure positions in the future will be those who demonstrate exceptional talents in examination scores. research, and personal attributes including mastering written and spoken English.

e) Before qualified physicians invest large personal and financial resources in the USMLE, these factors should be carefully reviewed.

f) Note that in 1996 over 7,000 qualified IMGs, a majority of whom interviewed personally in training programs in the U.S., were unable to find a position.

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Address: Karan Nagar, Srinagar Kashmir-190 009.

Date & Birth/Place: 16th July 1931, at Skardu (Gilgit)

Basic Education:
-CMS Central High School Fatah Kadal
-Interscience Examination East Punjab University

Medical Education:
MBBS; B.J.Medical College Ahmadabad (1954)
F.R.C.S. (Edinburgh) - (1959)
F.I.C.S. (USA) - (1968)
F..C.I.C.D. (Rome) - (1971)
F.I.M.S.A. (New Delhi) - (1981) as a founder Fellow).

Appointments:
Residency and Registrarship
-V.S.Hospital (Ahmadabad);
-S.M.H.S. Hospital Srinagar;
-Coventry and Warwickshire Hospital Coventry England.
Lecturer Anatomy - (2.6.1959)
Assistant Professor Surgery - (30.8.1961)
Associate Professor Surgery (13.9.1964)
Professor of Surgery (17.11.1967)
As Head of department Surgery (11.8.1972)

Research Projects conducted under his guidance on:- During this period research was conducted in department on Kangri Cancer, Needle Biopsies of Prostatic Disease, Tuberculosis of Gall bladder, Echinococcus cysts in unusual sites like Abdominal muscles, Pancrteas, Omentum etc. Surgical Complications of Ascariasis in Kashmir, Chemical studies of gall stones. About 27 publications in different Indian and International Journals were published.

Academic jobs:- During this period, has also worked as Inspector of Indian Medical Council, in addition to having worked as Examiner for MBBS and MS of Universities of Kashmir, Amritsar, Delhi, Patiala, AIIMS, Aligarh, Gujrat and National Board of Examinations (MNAMS) :

Memberships:- Member of Indian Association for Advancement of Medical Education, Life member of Indian Association of Surgeons, Member of Northern Chapter of Association of surgeons. Has worked as Head of department Surgery, Administrator SMHS Hospital, officiated as Principal Medical College, Srinagar.

Retirement:- 1.10.1982 (Premature)

Present job:- Senior Consulting Surgeon, (especially Gastroentrology surgery in private sector).

The Editor feels a great pleasure to complete a brief interview with my teacher and eminent Surgeon of the country :

(Q) Sir, what gives you great pleasure/satisfaction during your excellent services in the state till date ?

(A) The greatest pleasure has been to witness the rise of my students in this country and other countries. The greatest satisfaction has been to be able to be with my patients of different generations, inspite of local disturbed conditions. To be a silient spectator to the elevation of one's students to the Principalship of different Medical Colleges in the state and outside, to the posts of Heads of department of Surgery and unit Heads, gives me very great pleasure and tranquil gratitude.

(Q) What is your message for our young doctors?

(A) My message to my younger colleagues is very simple "Do your assigned job sincerely and do not bother about the results".

(Q) As a good citizen of valley, while continuing

to love and serve your people physically here under difficult situations, What is your message for Kashmiri people who are here or abroad ?

(A) My wish is that peace may once again prevail in this beautiful Motherland of Kashmir. Only those who have lived here for the last decade continously know as to how much we all have suffered in multiple ways. May God grant us once again tranquil and peaceful Existance.

(Q) A word about your family ?

(A) As far as my family is concerned, I have been married (as a medical student) to Mrs Sheila Bhan since 1951. My wife has been a great source of encouragement, boldness, truthfulness and above all a faith in spritualism. I have two sons, the elder Ravinder B.E. Hons. is great scholar & a appreciator of Kashmiri Poetry & folklore & is managing proprietor of Ravimech, who have the greatest no. of recorded Kashmiri audio cassettes. The younger Anil, M.S. M.Ch is cardiothoracic surgeon at AIIMS New Delhi, and his love for Kashmir & Kashmiris is examplary. I have a lovely daughter Savita (Wakhloo) B.E. (Hons) stationed at Jamshedpur along with her husband Bharat Wakhloo, Senion Manager with Tisco. They both are Mechanical Engineers turned Business management authorities. I have four grand children Vikrant and Ambika Bhan, Gobinda and Sapmada Wakhloo. May they all live in peace and as true Kashmiris.

From:
Prof. M.S. Misger.
MBBS ; (Hons), M.S.; FICS
Head of department of Surgery,
Govt. Medical College, Srinagar.

"The precious debt of learning is a debt that is difficult to pay, only gratitude can be felt. I feel highly previleged in expressing my deepest sense of gratitude and indebtedness to my venerated teacher and renounced surgeon Prof. B.M.Bhan, MBBS, FRCS, FICS, Ex-Professor and Head department of surgery, Govt. Medical College, Srinagar. He is well known for his enlighted guidance, constructive criticims, constant encouragement, generous help and unreserved co-operation in every field of life. He has always broadened our horizon of thought in the progress and up lift of our department to the present status. It is difficult to forget his most gifted and affactionate attitude towards our department. He is the best teacher & excellent Gastrointestinal surgeon in country.

(Prof. M.S.Misger)

From Editor,

Prof. B.M. Bhan is itself a domestic name for people of valley, a disciplined and noble teacher for his students, an honest and able Administrator for his subordinate staff, a man with simple habits, God fearing and religious man. He is the only emenient Kashmiri doctor, from minority community, who trusted in God and his people of valley and is living as well as serving them here, although he had facilities to live in any part of India. May God give him a long life to serve his people and family.

G. M. Malik

Dr. B.M. Bhan (As Lecturer Anatomy) seen with 1st batch of Students & Staff Medical College Srinagar (1959)

Meet the Professor from Basic Sciences

Dr. Mohd. Aslam M.S.

Place of Birth: Srinagar (J&K)

Educational Qualifications:
MBBS Vikram University, Ujjain 1960
M.S. University of Kashmir 1970.

Date of Joining G.M.C: (1962)

Present Appointment:
H.O.D. Anatomy, Acharya Shri Chander College of Medical Sciences & Hospital 147-A, A/D, Gandhi Nagar, Jammu (Tawi)

Past Appointments:
- H.O.D. Anatomy JVCMS, Srinagar
- H.O.D. Govt. Medical College Srinagar (1982-81)
- Associate Professor Alfateh University Libya (1979-81)
- Professor and Co-ordinator of Anatomy Imperial Medical Centre Iran - Tehran (1977-79)
- Associate Professor & Head University of Basrah (1975-77)
- Visiting Scientist University of Basrah (1971-73)

Examiner for Undergraduate/P.G: (MBBS & MS) University of Kashmir, Jammu, Punjab, Indore, Delhi, Aligarh, Lucknow, Basrah, Libya, Iran etc.

Paper Setter:
For National Board of Examination and Union Public Service Commission.

Publications:
Over 25 in National/International Journals including Recent Advances in Anatomy

Membership:
- Founder Member of Afro-Oceania Congress of Anatomist
- Life Member Anatmotical Society of India
- Inspector for MCI for MBBS, MS (1986-87)
- Member of a Committee to lay Salybii in Medical Schools in Iraq
- Selection Committee of Aligarh Muslim University

President:
Anatomical Society of India 1987

Special Interests:
Reproductive Biology

From Editor:

Prof. Aslam is a renowned teacher, who has produced hundreds of MBBS students & dozens of postgraduates (MS) in anatomy.

I remember him as a student, we used to be more afraid of him than Gray's anatomy, but everyone liked his simple teaching as well as examinership.

He would always dominate discussions in college faculty meetings like an opposition leader, enjoys healthy criticism even of his close friends. He never keeps anything secret & speaks whatever comes to his mind, even if it means loss of his friends. Always found him in a happy mood with young ideas & always looking like a young man 90's.

Although he comes from a high class business family but most of his family members are in medical profession. Although I found him a clear hearted teacher, colleague & friend, but there may be some people who will disagree with me, but that can happen with any person who has such a long carrier in this profession.

Obituary

1. Prof. M.S. Malik (1938-1996)

Dr. Mohammand Shafi Malik. Prof. of Surgery Govt. Medical College Kashmir (india)

Date of Birth 19-06-1938 (Bhadarwah) Jammu. Primary Schooling in Bhadarwah. Parents Expired while he was in primery school Graducation in Jammu. (His borthers brought him up and looked after his studies.) Did his MBBS from Shah Medical College Jamnagar (Shurashtra) in 1950-60, joined service in 1964 in Jammu, completed postgraduation from Medical college Amritsar. in 1967. He was appointed as Asstt. prof. in Medical college Srinagar. in 1975, as Associate Prof., and in 1988, as professor and was head of the Surgical Unit II till date and was due to retire in June 1996. Besides this he went to Foriegn assignment in IRAN for sometime.

He was going to take overfull time principal/Dean of Prime Medical College (private) after retirement. On 18th May, he died in a tragic accident while shopping with his family. He has left behind one son, 3 daughters & wife (Dr. Bilquis. Gyneacologist) People from all walks of life condoled his death including Medical College teachers Associations of Srinagar & Jammu. Employees Unions of SMHS Hospital. Prinicpal & Staff of Medical College & Prime Medical College. Rich tributes were paid to the departed soul.

May God give peace to his soul.

(Compiled by Prof. M.Ashraf)
HOD Radiology & President

2. Dr. B.A. Bhat

Dr. B.A. Bhat was assasinated at Tral (Noor Pora) on 7 June 1996 at age of 50 yrs. He was a Senior Medical Officer & a social worker. He has left behind 4 children. Rich tributes were paid to the departed soul by all people.

May God give peace to his soul

3. Mr. Abdul Majid Malik (Younger brother of Editor)

He was assasinated at his village Handwara on 11th May 1996, at age of 32 yrs. He has left behind 4 Children. People from all walks of life condoled his death & rich tributes were paid to his soul. Dr. Malik & his family pay thanks to all people who visited our house at Handwara & Srinagar on this sad occasion. Also thanks to all friends & colleagues from UK, USA & Saudi Arabia for their wirtten/Telephonic condolence messages send to us. Besides my thanks to Faculty of SKIM.S. Medical College Srinagar, Prime Medical College, Hussain Medical College. JV Medical College, Teachers Association, Local press/for their sympathetic messages send to our family. May God give peace to his soul.

Dr. Malik

MODERN ENDOSCOPY CENTER, JEHLUM MARKET, BATAMALOO, SRINAGAR.

Communications to Editor

Dear Dr. Malik,
Thank you very much for sending copies of JK Practitioner to the editiorial office of Scientific Communications. The contents proved most interesting. I wish you every success with JK Practitioner.
Kind regards

Scientific Communications (NZ) Ltd
John O' Brien, Editorial Director
Argosy House, 2nd floor, 87 Victoria
Street, Newzealand

Dear Dr. Malik,

Aslaamalaikum

I would like to congratulate you on your efforts in the publication and progress of the J.K. Practitioner. The spring edition was refreshing and the outline of the first group of graduates was good reading. It is certainly encouraging to see the first journal of our alma-mater and I look forward to receiving the next issue. Thanking you.

Sincerely
Tanveer Mir, M.D., F.A.C.P
Associate Program Director, Department
of Medicine Chief, Division of Geriatrics
Assistant Professor of Medicine
School of Medicine State University
of New York at Stony Brook

Dear Dr. Malik,

Asalamalaikum.

I am pleased to note the progress that JK Practitioner has made in a short period of time. I would like to congratulate you on your personal interest and endeavor in this difficult centure. I am enclosing a brief case-report for the journal. I will try to contact the alumni of our Medical College to render you some help in this difficult task. If I may be of any help please let me know. Regards to all at the college.

Sincerely,
Parvez A. Mir. MD
Attemdomg Physician
Pulmonary & Critical Care Medicine
Wyckoff Heights Medical Center
Wyckoff Heights Division
374 Stockholm Street
Brooklyn, New York 11232

Dear Dr. Malik,
It was a pleasure to go through the contents of present issue of the JK-Practitioner. The getup of Journal has improved and I wish you good luck in your future endeuvours & must congratulate you. Other friends who have received the Journal here are of the same opinion. I was sorry to know about the sad demise of prof. Beg & prof. I.P. Kohli, whom I had known very closely. I shall write some articles for the Journal very soon.

From Dr. G.Q. Allaqaband FRCP
Consultant Deptt. Cardiology
Wordesly Hospital, Stream Road,
Stourbridge, DY 8, U.K

Dear Dr. Malik,
Thank you for your kind letter dated 27th January 1996 which I received only yesterday. I have noted with interest that you have published our articles in the JK-Practitioner. but unfortunately I have not received any copies of these which I would very much appreciate if you could send along with your next issue.
My regards and wishes to you and your family.

Sincerely,
Dr. M.A.Al Karawi
Director Department of Gastroenterology
Ministry of Defence and Aviation
Medical Services Department
Riyadh Al Kharj Hospital Programme
Riyadh Armed Forces Hospital
P.O. Box 7897 Riyadh 1159
Kingdom of Saudi Arabia

Special Messages & Communications from Eminent People will be Published in Coming Annual Issue.

Information to Authors

The JK - PRACTITIONER Journal will henceforth receive and consider for publication, manuscripts prepared in the Vancouver Style (see Lancet : Feb, 24, Page 429, 1979) and manuscripts should be addressed to:

Dr. G M Malik, Chief Editor
JK Practitioner.
P.O. Box 884
GPO Srinagar - 190 001, J&K (India)

Manuscripts will be received for possible publication with the understanding that they have not been published, simultaneously submitted, or already accepted for publication by another journal. The corresponding author shall have to furnish a declaration to this effect on behalf of all the authors. Manuscripts should be submitted in triplicate including the illustrations. Authors are encouraged to submit manuscripts on diskette (properly labelled) in Dos or Windows format in any popular word processing package. Three print out copies of the papershould be supplied as well.should be supplied as well.

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Text - The text of observational and experimental articles is usually but not necessarily divided into sections with the headings; Introduction, Methods, Results and Discussion. To clarify their content, especially in the results and discussions sections, long articles may need subheadings.

Acknowledgments - The persons who have made substantive contributions to the study can only be acknowledged. It is the author's responsibility to obtain written permission from the persons whose name have been acknowledged because readers will infer their endorsement of the data and conclusion.

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Srinagar, Kashmir,India
P.O.Box 884, Pin Code 190 001

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Computer Execution
Arshad Sofi.
At :- **Skyline Computers**
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Editorials:

-Sepsis Syndrome Therapy 206 Farooque A Khan, R Bashir
-The Chronicle of Growth Hormone Insensitivity 209 AH Zargar, BA Laway, SR Masoodi, FA Sofi, AI Wani
- Postmenopasual Hormone Use-Time for Concensus 212 Bilquees Jamila, N Firdous, Zaffar Abbas

Guest Article:

-Fulminant Hepatis Failure 214 PK Garg, Rakesh K Tandon

Clinical Reviews:

-Application of Laser in Sugery 220 NA Wani
-Ischaemic Strokes:- An Update 222 PA Shah, A Nafi

Mini-Reviews:

-Gastrointestinal Mucosal Protective Activity of Aluminium Ion-A 226 GN Mir
-Tobacco & the Integument 229 GH Hajini, Pervaiz Shah, Iffat Hassan, Abdul Ahad

Original Articles:

-The Pattern of Child Psychiatric Disorders in Kashmir 233 Mushtaq A. Margoob
-Tumor Pattern in Undescended Testis 237 ND Choudry, AR Khan
-A Study of Trace Elements, Copper and Zinc in Patients of Head, Neck and Oesophageal Malignancies 241 SJA Bihaqi, M Latif, Sheikh Abdul Ahad
-Fine Needle Aspiration Cytology as an Initial Tool in Evaluation of Head and Neck Masses 245 MA Darzi, NA Chowdri, MA Bhat, GM Jan

Experimental Medicine:

-Lithium and Kidney 249 MS Shah, Hassan G

Case Reports:

-Clouston Syndrome 251 Masood A Qazi, S Manzoor, Aftab Ahmad
-Late Metastatis of Malignant Melanoma to Bladder 252 AA Bhat
-Recurrent Ameloblastic Fibroma with Atypical Presentation 253 Naseer A, MA Darzi, Shabir Iqbal, ZA Khan

Special Articles:

-"Islamic Teachings & Cancer Prevention" Part II - Alcohol and Cancer 255 MA Albar
-Glimpses of Research in Medicine in the Valley for the Last Two Decades 260 Mohd. Yousuf

Imaging Today:

-Magnetic Resonance Imaging & Proptosis 263 S Sajad, Bashir A, AR Nasti, Tejit Singh
-Ultrasound Pictures of GIT Ascariasis 265 GM Malik, S Kadla
Clinical Images: 266 AH Zargar, SR Masoodi, BA Laway, S Iqbal

Doctor and Patient Relationship:

-Medical Ethics 267 S Amin Tabish

Practitioners Column:

-Migraine - New Horizons 273 Abdul Salam

Students Page:

-Tips on Critical Care Medicine 276 A Yousif
-Tips on Advances in Hepatology 278 Roger William
Journal Scan: 279 Zaffar Abbas

Overseas diary:

-The New Undergraduate Curriculum for the University of Wales College Of Medicine 280 AM Mir

Meet the Professor:

-Dr. G.N. Mir 283
Obituary: 284
Communications to Editor: 285
Index Vol. 3 (Nos. 1-4)

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-> **Cardiac Pacemakers**
-> **Abdominal Hystrectomy**
-> **Hair Cosmetics**
-> **Pulmonary Hydatid Disease (SKIMS experience)**
-> **Diabetic Foot**
-> **Medicine: Pride and Prejudice**
-> **Serum Calcium Levels in Pulmonary Tuberculosis**
-> **Psoriasis and Pencillin**
-> **Cholecystectomy and Appendicectomy or Cholecystectomy alone**

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From the Editor's Desk

JK - Practitioner

Aims and Scope

It gives me immense pleasure to present before the readers. this annual issue. We have been able to continuously publish three volumes till now and our efforts will not lag behind to make it as one of the foremost source of medical information for doctors.

JK Practitioner is a quarterly multidisciplinary medical journal being published since Oct. 1994 from Kashmir. Emphasis is placed on matters relating to medicine in this country in particular. although articles from anywhere in the world are given due importance. We welcome high quality papers dealing with various aspects of clinical. academic. investigative and social medicine.

I am greatly thankful for the guidance. patronage and help provided by my colleagues. here and abroad. I acknowledge and appreciate their efforts. in this regard I would particularly like to thank Prof. Khan (USA). Prof. P.K. Shah (USA). Prof. U. Koul (Delhi). Prof. M.A. Mir (UK). Many eminent teachers and clinicians of the valley like Prof. S.N. Ahmad Shah. Prof. S.Z. Ahmad. Prof. Allaqaband. Prof. Girja Dhar. Prof. Wazira Khanam and Prof. Khuroo have put in their bit to improve the quality of the journal. besides the editorial & advisory board members.

This journal publishes original articles. case reports. letters to the editor. communication. reviews. guest articles. besides interviews of notable doctors. I am thankful to all the esteemed doctors who have been contributing for the journal from all over India and abroad and hope they will continue to do so in future.

The editorial board wishes to minimise the delay between manuscript submission and its acceptance for publication and accordingly authors are requested to follow carefully the instructions set out under "information to authors".

Our readership is increasing day by day. We hope this journal will continue to promote medical education to our doctors and we will try to overcome the deficiencies. as there may be. in the future issues.

Looking forward to your valuable suggestion and constructive criticism.

G.M. Malik MD; FACG
Editor-in-Chief

The editorial team of JK Practitioner/ Medical College Teachers Association/ Faculty congratulates the Principal & the faculty of Govt. Medical College Jammu on their Silver Jubilee year deliberations 1996.

SEPSIS SYNDROME THERAPY

Status of Anti-Endotoxin and Anti-mediator Trials - 1996

JK Practitioner 1996: 3:4 206-208

"Where We are, Where We are Going"

Sepsis is the thirteenth leading cause of in hospital mortality in the United States[1], an estimated five hundred thousand patients develop sepsis yearly with an estimated one hundred thousand deaths. Despite tremendous advances in antibiotic therapy and in the care of critically ill patients, the mortality rates for sepsis have remained high ranging from 20-40% and 40-70% for those in septic shock.

Great progress has been made recently in identifying the biochemical mediators associated with infection and its complications like multi-organ system dysfunction syndrome (MODS), hypotension and death. The pathophysiology of this syndrome can be compared with a biochemical "panic attack", in which large amounts of various cytokines are released into the circulation in response to infection. These cytokines are responsible for progression of sepsis syndrome to septic shock and death.

The traditional treatment of Sepsis Syndrome consists of the use of anti-microbial agents, elimination of the source of infection, hemodynamic support and treatment of various complications. Recently several multi center, randomized placebo controlled, double blinded trials have been completed with an objective of evaluating the efficacy and safety of various drugs that modulate the effects of cytokines or endotoxins in patients with sepsis syndrome. The purpose of this brief report is to provide a status report on the various newer modalities used for the treatment of sepsis syndrome and septic shock.

The inclusion criteria for these studies were based on definitions of ACCP Consensus Conference in 1991, which are as follows:-

Criteria for Defining the Spectrum of Disorders Associated with Sepsis

Sepsis: Clinical evidence of infection plus all of the following:

1. Tachypnea (Respiration greater than 20 breaths per min or if mechanically ventilated more than 10L/min)
2. Tachycarida (a heart rate more than 90 beats/min)
3. Hyperthermia or Hypothermia (core or rectal temp less than 35.6°C or 96°F, oral temp more than 38.4°or 101 °F.)

Sepsis Syndrome: Clinical diagnosis of sepsis as outlined above plus one or more of organ hypoperfusion:

1. PaO_2/F_IO_2 less than 280 (in absence of pulmonary or cardiovascular diseases)
2. Metabolic acidosis or elevated lactate levels
3. Oliguria (Urine output less than 0.5 ml/min for at least one hour)
4. Acute alteration in mental status.

Sepsis Shock: Clinical Diagnosis of sepsis syndrome as outlined above plus Hypotension (systolic blood pressure below 90 mmHg or a 40 mmHg drop below the baseline systolic blood pressure) that for more than one hour despite adequate fluid challenge.

The various modalities of therapy that are being investigated are:

1. Anti-Endotoxin antibodies
2. Anti-mediators
3. Modulation of Neutrophil Function 4. Nitric oxide Synthetase inhibitors.

The later two modalities are still restricted to animal studies, however anti-endotoxin and anti-mediator therapies are being extensively investigated in large clinical trials

Anti-Endotaxin Antibodies:

Anti-endotoxin antibodies can be polyclonal, e:g J5 anti-serum, or Monoclonal eg. E5 (Murine IgM), HA-IA (Human IgM) and are produced against J5 mutant of E.Colli[7]. In the first clinical trial of J5 anti-serum, 191 patients with gram negative infection were enrolled. The results showed that overall sepsis related mortality was reduced from 39% to 22% A retrospective subgroup analysis revealed that those patients, requiring vasopressors for greater than six hours, the mortality was reduced significantly from 77% to 44%. Unfortunately subsequent five such clinical trials failed to show such a benefit.

Four hundred and sixty eight patients with gram negative infection were enrolled in a clinical trial using E5 anitbodies. This showed no survival benefit compared to placebo, however, a subgroup

Received & Accepted August 1996

analysis revealed an improved survival in patients with documented gram negative infection without refractory shock. Another clinical trial of 847 patients without refractory shock amongst which 530 patients had documented gram negative infection, showed no survival benefit. A met analysis of both these trials showed an improved survival by patients with organ failure but without refractory shock. Based on this hypothesis a third E5 clinical trial is underway.

540 patients with evidence of gram negative infection were enrolled in clinical trial of HA-IA versus placebo. Results of this trial showed no overall survival benefit, however, a retrospective subgroup analysis showed a statistically significant decrease in day 28 all-cause mortality in patients with gram negative bacteremia. Based on this the drug was approved for marketing in Europe. A second major clinical trial of HA-IA was started in 1992, and an interim analysis a year later, showed a statistically significant increase in mortality compared to placebo. Hence the study was terminated prematurely and the drug was withdrawn from the European market.

The more potent Anti-Endotoxin agents are still being investigated in their initial clinical trials and they include Bactericidal permeability-increasing (BPI) Peptide, Polymyxin B derivatives, high, low and very low density lipoproteins and nontoxic derivatives of lipid A. The later one includes lipid X, lipid IVa Monophosphoryl and diphosphoryl lipid A. Several case reports of using polymyxin B immobilized fiber (PMX-F) in blood filters, which selectively bind the endotoxin have been published.

The possible reasons for the lack of efficacy of these anti-endotoxin agents appears to be: (1) The weak ability of these agents to block the activating effects of endotoxoins on macrophage release of proinflammatory cytokines. (2) Endotoxin may not have a fundamental pathogenic role in human septic shock. (3) The design flaws of previous clinical trials may have led to type II error. (4) Study entry criteria might not be selecting patients most likely to benefit.

Anti-mediator therapies:

Anti-mediator therapies have generated great interest amongst internists and critical care researchers interested in sepsis syndrome. The success of these therapies will herald an era of new modality of treatment, perhaps similar to the advent of Antibiotics. Anti-mediator therapies are supposed to be effective against both gram negative and gram positive infections. These therapies include IL-I receptor antagonist (IL-IRa), **Monoclonal antibodies to tumor necrosis factor alpha (TMNF MAb), Soluble receptors for TNF-alpha, Prostaglandin inhibitors like NSAID and steroids, Bradykinin antagonists, platelet activating factor antagonists etc.**

An open labeled study of 99 patients with sepsis syndrome showed a decrease in day 28 all-cause mortality by IL-IRa versus placebo. This was tested in another multicenter, double-blinded, randomized placebo-controlled trial of IL-IRa which enrolled 893 patients. This study showed no differences in survival between the placebo group and the IL-IRa group. However,a retrospective subgroup analysis, based on the "Risk prediction model", showed a statistically significant improvement in survival of patients whose predicted risk of death was greater than 24% .This hypothesis is being investigated in another ongoing clinical trial.

The Veterans Administration systemic sepsis cooperative study group and methyl prednisone in severe sepsis study group conducted two randomized, placebo-controlled double blinded trials using high dose glucocorticoids in patients with sepsis syndrome and septic shock. Both these trials showed no survival benefit. However, clinical trials using cyclo-oxygenase inhibitor like Ibuprofen are currently underway.

The possible role of Tumour necrosis factor alpha as a central mediator of systemic inflammatory response syndrome (SIRS) has generated significant interest in critical care arenas, because of the therapeutic potential of its inhibitors. Recently reported phase II double blinded, randomized trial of recombinant soluble TNF-alpha receptors in 141 patients with sepsis syndrome, showed that day 28 all cause mortality was not different for low dose (0.15 mg.kg 30%) and placebo group (30%) but patients who received a high dose (1.5 mg/kg 53%) had a worse outcome (P=0.016)[4]. Hence future clinical trials will use very low doses of soluble TNF alpha receptors.

NORASPET-I (North American Sepsis trial) using monoclonal antibodies to TNF alpha, results were published recently[8]. Nine hundred and thirty one patients with sepsis syndrome were enrolled, and prospectively stratified into shock and nonshock groups. The results indicated that there was no improvement in day 28 all-cause mortality. However, there was a trend towards improvement in patients with septic shock, it was statistically significant on day 3 post-infusion, but did not attain statistical significance on day 28. There was no statistically significant difference in mortality between 7.5 mg/kg dose versus 15 mg/kg dose of TNF [illegible] A second phase II trial called NORASEPT-II is currently in progress and investigates the role of TNF MAb in treatment of patients with septic shock. The other clinical trial which tested TNF MAb was INTERSEPT-I (International Sepsis Trial), which showed no survival advantage, however there was a decrease in duration of shock.

Results of Phase II/III Double blinded, placebo controlled, randomized, multi-center clinical trial of Bradykinin Antagonist (CP 0127)) were recently reported. This prospective trial evaluated 3-day infusion of three different doses of the drug versus placebo in 500 patients with SIRS and presumed Sepsis. There was no statistically significant effect on mortality, yet there was a trend towards improved survival during and immediately after infusion. Studies are underway to evaluate 7 day infusion of Bradykinin antagonist versus placebo. Another important anti-mediator PAF Antagonist - BN52021 was tested in a double blinded placebo-controlled trial involving 262 patients. It showed no overall improvement in survival, however, subgroup analysis revealed that in a subgroup of patients with documented gram negative infection the mortality was reduced from, 57% to 33% in the treated group.

In addition to the equivocal results of the clinical trials, the treatment with these agents is expensive which has several implications in the cost conscious environment. For example three years ago a single dose of HA-IA cost approximately $3,700. in Europe.

Conclusions

Despite the impressive experimental findings of improved survival in animal models with anti-mediator and anti-endotoxin therapies, clinical trials to date have shown no clear cut benefit, the only exception being NORASEPT-I which as mentioned above showed a trend toward improved survival but did not achieve statistical significance.

Clearly the animal models for sepsis, which use endotoxin or bacterial insults in previously healthy animals, differ markedly from the clinical situation where infection usually has no well defined starting time and occurs in patients with pre-existing medical problems. In addition, this disparity forces a rethinking of the paradigm that a "single mediator can play a central role". Even modest improvements in the outcome of various subgroups necessitates defining certain biochemical and clinical markers which will better identify the necessity for using a combination of various modulators to achieve meaningful decrease in mortality.

F. Khan MB, MACP, FCCP
Prof. and Head
R. Bashir Senior Resident
Deptt. of Medicine
Nassu County Medical Center
East Meadown NY: and
State University at Stony Brook (USA)

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The Chronicle of growth Hormone Insensitivity Syndrome

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Introduction

Growth retardation is a common clinical problem. Failure to thrive in children may be due to a number of factors, growth hormone deficiency (GHD) being one of them. Following it's secretion by the anterior pituitary gland, growth hormone (GH) must be bound to its receptor for normal regulation of growth and metabolism to occur. Inspite of normal or high concentration sometimes, GH fails to exert its biological actions. The resulting syndrome has been variously termed as Laron dwarfism or growth hormone insensitivity syndrome (1). Unlike children with GHD treatment with GH does not raise their insulin like growth factor - I (IGF-I) concentration or stimulate growth (2,3)

Clinical characteristics

Pregnancy and delivery are usually normal through some parents have noticed affected children at birth because of their facial features, sparse hair, small hands and feet and hypoplastic finger nails. Associated malformations including short fourth metacarpal, clinodactyle brachymesophalangy, strabismus, cataract, nystagmus, aortic stenosis, undescended testes, cleft lip, ptosis with facial asymmetry have been reported (4,5). Blue sclerae have been reported in Israel and Ecuadorean patients (6). Hypoplasia of the bridge of nose with prominence of forehead is a uniform feature in children but may be less apparent is some children and in many adults (4). There is a high rate of decay of primary teeth because of their long retention, thus the problem is rarely seen before 5 years of age but occurs in approximately 75% of older children.

Biochemical Characteristics

Patients with Laron-type dwarfism are clinically indistinguishable from those with isolated (GHD) yet have high circulating GH concentration associated with an inability to generate endogenous IGF-I. Normal binding of GH to various antisera used for radioimmunoassay of GH have been demonstrated (7). Jacob et al demonstrated that GH from seven patients with Laron-syndrome behaved normally in a radio-receptor assay suggesting that defect may be at the receptor level (8). In 1990, a remarkable concentration of growth hormone receptor deficiency (GHRD) was reported from an inbred Caucasian population in a single province in southern Ecuador (7). Among European patients IGF-I generation tests have been used to distinguish GHRD from GHD with serum IGF binding protein - 3 (IGFBP - 3) response being more discriminatory than those of IGF - I (9). A sluggish response shown by some patients suggest that there may be a spectrum of GH resistance from partial to absolute in the populations. Although most patients have very low or undetectable levels of GHBP substantial numbers have measurable levels and some have normal levels (10,11). This has been thought to suggest genetic heterogeneity.

The GH receptor gene has been localized to human chromosome 15 (12) and is over 40 times larger than the GH gene. The cDNA for GH receptor have been cloned from a variety of primates including human, rabbits and rats. The extra cellar domain of the GH receptor is normally found in serum as GHBP. The binding affinity of GHBP for GH is about the same as that of intact GH receptor (13). The absence of GH receptor activity and GHBPs in Laron syndrome suggested that GH receptor and GHBP might be identical or closely related (14). Point mutation in the mutant growth hormone receptor gene has been described in patients with Laron syndrome (15). Elucidation of the molecular mechanisms responsible for Laron syndrome have beem rapid and promises to be helpful in genetic councelling affecting families.

Historical and Geographical Perspective

Laron et al described the siblings with hypoglycemia and other clinical and laboratory signs of GHD with an abnormally high concentration of immuno reactive serum GH (17). In 1968, the Israeli investigators described 20 patients noting a high degree of consanguinity and possible autosomal recessive inheritance. The condition appeared only in oriental Jews (17). The first patients reported from outside Israel by Merimee et al in 1968 was a 30 year old man who demonstrated no metabolic response to exogenous GH (18). In 1969 Daughaday et al reported defective sulfation factor activation by GH in six of the patients with Laron syndrome from Israel (19).

In 1990, a remarkable concentration of GHRD was reported from an inbred Caucasian population in a single province in Southern Ecuador with paucity of affected males (20). Subsequently a clinically and biochemically similar population from a neighbouring province was reported (21). Till recently only seven patients with Laron dwarfism had

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been reported from Indian subcontinent (22-24). In one study from Bombay 11% of two children with possible growth hormone deficiency were reported to have GH resistance and there was a marked predominance of familial cases (25).

We reported an overt case of growth hormone resistance in 1990 (26), this was subsequently followed by a report of sporadic and familial growth hormone insensitivity syndrome (27,28). Consanguity was common in our patients. Our patients had characteristic cherubic facies, prominent forehead, depressed nasal bridge and a small mandible.

Therapeutic modalities

Our understanding of the mechanism of action of GH has over the last decade lagged considerably behind that of the other hormones. Cloning of a GH receptor cDNA, however has made possible to study this molecule using in vitro systems and to analyse at the molecular levels patients with GHRD (2,16,29).

Administration of IGF-I to patients with Laron type dwarfism seems to have a beneficial effect on growth similar to that observed with long term administration of GH in children with GHD (30). The initial clinical trials with IGF-I are aimed at assessing its potential for promoting growth in children with GHRD (31,32). Such systemic use of IGF-I involves relatively low doses of IGF-I that raise the circulating therapy relies on resorting the psychosocial role of circulating IDF-I. In the first report of injection of IGF-I into humans there were severe hypoglycemic reactions (33,34). However, there is no doubt that IGF-I will enter the therapeutic era, the dosage and the therapeutic regimen will depend on the endpoint desired.

A.H. Zargar, B.A. Laway, S.R. Masoodi,
F.A. Sofi, A.I. Wani
Department of Endocrinology
Institute of Medical Science,
Soura Srinagar Kashmir

Correspondence:
Dr. A.H. Zargar
Associate professor and Head
Department of Endocrinology
Institute of Medical Sciences,
Soura, Srinagar, Post Bag No. 27
Srinagar - 190 011 India

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Postmenopausal Hormone Use - Time for consensus

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The importance of postmenopasual hormone (PMH) use in relieving the unpleasant symptoms of menopause (hot flushes, vaginal dryness, insomnia, headache, dyspareunia, poor concentration and memory, irritability and depression) is well established and widely accepted.[1,2,3]. What is equally well documented is the role of this therapy in prevention of postmenopasual osteoporosis. Oestrogen replacement not only prevents the accelerated bone loss which accompanies menopause but also reduces the subsequent risk of fracture. The positive effects of oestrogen on bone density which is dose related, is way ahead in what is achieved by non-hormonal alternatives (calcius, calcitonin, bisphosphonate) in preventing osteoporosis in these women.[4]

The same kind of certainty cannot be ascribed to the impact of longterm hormone replacement on the cardiovascular status. PMH use in ischemic heart disease, stroke and thrombosis is still mired with controversy. The Framingham study[5] did suggest an adverse effect but most of the subsequent studies failed to show the adverse relation between coronary artery disease and oestrogen therapy. Stampfer and Colditz[6] in a meta-analysis of 31 controlled studies indicated that the risk of CAD with PMH use was significantly reduced. In a case controlled study of Psaty and others[7] which included 502 cases of postmenopasual women who had sustained a fatal or non-fatal MI and 1193 controls, also showed a decrease risk of MI associated with oestrogen use. Kendall ans Horton[8] in their review of recent advances in drug treatments have reported a study which included 933 postmenopasual women who had undergone coronary angiography. The occlusion scores in oestrogen users (154 in all) was significantly lower than in non users. A longitudinal study by Finucane and others[8] have concluded that PMH use was a protective factor against stroke incidence and stroke mortality in postmenopasual women.

The single factor which has slowed down the acceptance of PMH use as an established practice is the concern, apprehension and controversy regarding the possible risk association of such treatments with malignancy. (Breast ovary, enometrium). Dealing with the subject Fitzgerd[9] has forcefully brought out the point that fears over the increase incidence of gynecological and breast maligancies associated with oestrogen replacement are not justified and that women should not be denied treatment on these grounds. In fact they are of the view (with substantial backup) that women treated for early stage carcinoma of the endometrium and ovary have a better chance of survival when treated with replacement oestrogens. Hunt et al [10,11] have reported reduced mortality from breast cancer in patients treated with oestrogen.

Cyclic Progestogen with oestrogen may be an established practice with some kind of an insurance against development of endometrial hyperplasia due to unopposed oestrogen therapy but, cyclic bleeding, psychological and physical symptoms of progestogenic therapy remain a problem. This could be overcome by low dose substitution or by using new synthetic derivatives of norethynodrel[4]. Hystrectomy in postmenopasual women may completely avoid the need for progestogen. The new non oral oestrogen delivery (Transdermal patch), besides minimizing the thrombotic tendency (hepatic bypass) has a better hormone profile over long periods, ofsetting the minor disadvantages which sometimes call for discontinuation of therapy; (Skin irritation, adhesive problems in hot weather). Transdermal delivery systems are being developed which will minimize these effects. Results of using a combined oestrogen/progestogen patches is also available.[12] Subcutaneous oestradiol implatns have an advantage of less frequent administration (6 monthly), so better compliance. With a dose titrated at 25-50 mg (5-6 month) the chances of supraphysiological oestradiol level attaintment and tachyphylaxis is minimized.[4]

Hemminki and Sihvo[13] has an important and interesting viewpoint, in that, the prescribing practices of physcians in the United States regarding PMH use may be dictated on the advice given on oestrogen prescribing in the physicians Desk Referance, few standard books and other written sources. The study covered the material available to the physcians from 1945-1990, which in itself has been contradictory and variable intime and text. If this is true the authors feel that an unintentional bias may have gone in selecting patients for PMH use and this bias when translated in to various surveys may have underestimated the risk of breast cancer and overestimated the protective effect against fractures. The impact of this study on the cardiovascular disease, they feel is less clear.

It has been found that gynecologists are much more favourable towards hormone therapy than other physicians. Is the time not ripe for cardiologists, psychiatrists, neurologists and general

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physcians to sit, reflect, pause and emerge with a consensus, as oestrogen replacement has an important role in preventive medicine within their own practice. The report of the Women's health initiative (which includes clinical trails of HRT) may make the decision easier, but the results may not be available until about 2006.

Bilquees Jamila MD
Professor of Obstetrics and Gynaecology
Nighat Firdous MD
Registrar, Deptt. of Obst. and Gynae.
Zaffar Abbas MD, Deptt. Of pharmaoology
Govt. Medical College, Srinagar, Kashmir

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"Fulminant Hepatic Failure"

Pramod K Garg,; Rakesh K Tandon***

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Definition:

Development of encephalopathy and severe coagulopathy within 8 weeks of onset of jaundice without any evidence of pre-existing liver disease is termed as fulminant liver failure (FHF)[1]. The duration between onset of jaundice and appearance of encephalopathy considered for the diagnosis has however varied from 4-12 weeks. Recently, Williams et al have divided the syndrome into three categories depending on the time lag between onset of jaundice and encephalopathy: hyperacute liver failure - within 7 days, acute liver failure (ALF) between 7-28 days, and subacute liver failure between 28 days to 12 weeks.[2] The prognosis was best in hyperacute liver failure and worst in subacute liver failure. however, this definition leaves patients with development of encephalopathy between 12-24 weeks of onset of jaundice because definition wise chronic liver disease is diagnosed when the total duration of the disease exceeds 24 weeks. We, at the All India Institute of medical Sciences (AIIMS), New Delhi, use a different classification. We diagnose FHF when the onset of encephalopathy is within 4 weeks of jaundice. This is because more than 90% of our patients with FHF develop encephalopathy within 4 weeks. patients developing signs of liver failure in the form of ascites/edema with or without encephalopathy between 4-24 weeks of onset of jaundice are labelled as subacute hepatic failure (SAHF).[3] Although there seems to be some confusion regarding the duration of jaundice before encephalopathy sets in, it is clear that FHF represents severe acute liver injury resulting in vital loss of liver functions which causes a clinical picture of jaundice, encephalopathy and coagulopathy.

Magnitude of problem of FHF:

At the A.I.I.M.S., New Delhi, we treat about 40-50 patients with FHF every year which form approximately 7% of all admissions (about 600-700) per year in the department of Gastroenterology. FHF complicates the course of acute viral hepatitis during epidemics in 0.5 of nonpregnant patients and 2.4% of pregnant females. The problem is further magnified by a very high mortality rate which vary from 60-90% in different series.

Aetiology of FHF[4]:

The most common cause of FHF is viral hepatitis (table 1). Among the viral causes, hepatitis E probably accounts for the maximum number of cases in India followed by hepatitis B while in the western countries hepatitis B is the commonest cause. Hepatitis A rarely causes FHF while hepatitis C does not cause FHF. The incidence of FHF in hepatitis B viral infection is about 1%. The second most common cause of FHF is drug induced liver injury. Among the various drugs acetaminophen is the commonest culprit in UK while antituberculosis drugs such as isoniazid and rifampicin are in India. Other causes of FHF such as metabolic and vascular are distinctly rare.

Diagnosis:

Clinical features: In most cases Jaundice is preceded by a short prodrome. Encephalopathy develops at variable time interval. At times the patient is brought to the emergency department in a state of coma and the jaundice is detected by the physician. History of jaundice occuring in the neighbourhood or the community will point toward an epidemic of acute viral hepatitis. Alternatively, a history of hepatotoxic drug intake such as anti tuberculosis drugs may be available. Physical examination shows presence of jaundice, reduced liver span on percussion (normal 12-15 cm) and encephalopathy verying in grade from 1-4 (table 2)

Investigations:

Liver Function Tests: Serum bilirubin is raised in all patients with FHF, Very high levels of bilirubin (17mg/dl) in patients with FHF however, suggests bad prognosis and forms one of the criteia for selection for orthotopic liver transplantation (OLT). On the other hand, even a very high level of biliru-

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**Professor and head, Department of Gastroenterology, All Inida Institute of Medical Sciences, New Delhi.*
***Pool officer, Department of Gastroenterology, All India Institute of Medical Sciences, New Delhi.*

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bin in a patient with acute hepatitis does not suggest a propensity to develop FHF. The aminotransferases are raised and the levels may reach thousands but their levels bear no correlation with either the development of or outcome of FHF. Serum alkaline phosphatase is either normal or minimally raised. It is typically low in Wilson's disease. Prothrombin time is invariably prolonged and is a sensitive marker of severity of liver injury; it forms an important criterion for OLT.

Haematology: Total leucocyte count is normal or raised. Platelet count may be low.

Serum Biochemistry: Blood urea levels are low as urea is synthesised by the liver. High levels of bilirubin interfere with the measurement of creatinine and hence creatinine values may not be an accurate guide if there is associated renal failure. There may be profound electrolyte disturbances such as hyponatraemia, hypo/hyperkalaemia and hypocalcaemia.Arterial blood gas analysis may show a low pH especially in paracetamol induced FHF, a low pO2 (hypoxia) and a normal or low pCO2 due to hyperventilation.

Imaging: Chest radiograph may show signs of pneumonia or patchy infiltration or shadows as seen in ARDS. Ultrasonography and CT scan, although rarely required, may show presence of large areas of liver necrosis.

Histopathology: Liver biopsy is not recommended during FHF due to severe coagulopathy. However, histopathology of the liver done either post-mortem or after recovery shows massive necrosis and inflammation.

Serology: IgM antibodies against hepatitis A,B, D, or E virus may be positive. In many patients with hepatitis B induced FHF, HbsAg may not be positive because intense immune attack against the virus infected hepatocytes leads to clearance of the virus.

Differential Diagnosis:

1. Malaria: Cerebral malaria presents with encephalopathy and may be associated with mild jaundice due either to hemolysis or mild malarial hepatitis. At times severe malarial hepatitis can lead to jaundice and encephalopathy. There is a history of continuing high fever (unlike acute viral hepatitis wher fever subsides within a few days) and the liver is generally palpable. Spleen may be enlarged. peripheral blood film examination clinches the diagnosis.
2. Enteric fever: Acute enteric fever may cause encephalopathy and acute hepatitis. This is not classified as FHF since encephalopathy is not due to severe liver damage but due to enteric fever per se. Blood culture and widal test are helpful in the diagnosis.
3. Septicaemia: Sepis is one of the causes of acute liver dysfunction. It can lead to mild cholestatic hepatitis or at times cause severe liver damage. Many patients with septicaemia may present with fever, jaundice and altered sensorium. The diagnosis is made in presence of signs of sepsis in form of tachycardia, tachypnea, hypo/hyperthermia, raised TLC, and positive blood cultures, The liver span in normal. The prognosis is good if sepsis is controlled with effective antibiotics.
4. Acute fatty liver of pregnancy: In third trimester of pregnancy, development of jaundice and encephalopathy should raise the suspicions of acute fatty liver of pregnancy. The liver is not shrunken. Uric acid level may be arised. Serology is negative. Diagnosis can only be confirmed by a liver biopsy and staining the tissue for fat. Acute fatty liver of pregnancy is in fact a cause of fulminant hepatic failure.
5. HELLP Syndrome: In toxaemia of pregnancy patients may develop hemolytic anaemia (H), elevated liver (EL) enzymes due to acute liver injury and low platelets (LP)> These patients usually do not have severe coagulopathy, reduced liver span or deep coma. Signs of toxaemia are present.
6. Chronic liver disease: Many patients with chronic liver disease present with encephalopathy and jaundice. The presence of ascites is an important sign to suggest underlying chronic liver disease. past history suggestive of liver disease and chronic alcoholism may also be helpful in differentiation.

Complications of FHF:

(i) Cerebral edema: The most dreaded complication of FHF is cerebral edema. Cerebral edema i.e increased intracranial pressure (ICP: 25 mmHg) accounts for the majority of deaths in patients with FHF. It develops in 50-80% of patients who are in grade III-IV encephalopathy. The cause of cerebral edema is not very clear. Both vasogenic and cytotoxic mechanisms appear to play a role in its genesis[5]. Increased intracellular concentrations of osmogenic alanine and glutamine, which are products of ammonia, may lead to swelling of the cells. Clinical signs which suggest presence of cerebral edema are bradycardia, raised blood pressure to maintain cerebral perfusion, increased muscle tone. hyperventilation, Cheyne stokes breathing and decerebrate posturing. Papilledema is uncommon. ICP monitoring by extradural device is indicated in centres where transplant facilities are available.

(ii) Infection: Infection is the second most common killer in patients with FHF. In one study upto 90% of patients with FHF had bacterial and 32% had fungal infections.

iii) Gastrointestinal bleed: It occurs in as many as 50% of patients . Coagulogram is grossly deranged in patients with FHF since most of the procoagulant factors which are synthesised in the liver are low. In addition, there is platelet dysfunction and sometimes thrombocytopenia.

iv) Acute renal failure: Development of oliguria is an ominous sign. ARF occurs in about 50% of patients. it may be due to sepsis leading to acute tubular necrosis or due to hepatorenal syndrome.

v) DIC: In some patients, DIC may developed with resultant bleeding diathesis and may require transfusion of blood.

vi) ARDS: Respiratory embarrassment is common in patients with FHF. It may be due to cerebral edema, lung infection or ARDS.These patients require intubation and often mechanical ventilation.

(vii) Intravascular hemolysis: This may occur in those with preexisting G-6 PD deficiency or when the cause of FHF is Wilson's disease, Cola coloured urine and very high levels of bilirubin suggest the diagnosis. It requires blood transfusion and maintenance of good urine output. Urine should be alkalinised.

Treatment:

Medical Management: Medical treatment of FHF essentially consists of intensive monitoring and supportive therapy. Although, FHF carries a very high mortality, it is a potentially treatable disease and hence no effort should be spared in treating this problem.

Monitoring: Patients with FHF require careful round the clock monitoring because early detection of complications and timely intervention is the key to successful outcome. Ideally such patients should be admitted in ICU. Every hour their pulse, BP, respiratory rate an rhythm, temperature, muscle tone, papillary reflexes, grade of coma, and urine output should be charted. They should have a central venous line for pressure measurement. Serum electrolytes, blood sugar, TLC, prothrombin time and arterial pH and gas analysis should be done at least once and ideally twice a day.

Supportive Treatment: All patients with FHF should have a nasogastric tube, central line and urinary catheter (preferably condom connection in males if there is no urinary retention) placed soon after admission to the hospital. Maintenance of fluid and electrolyte balance is of utmost importance. Any excess of fluid will lead to or exacerbate cerebral edema. They should receive 10% dextrose drip throughout the day to prevent hypoglycaemia to which these patients are quite prone. Electrolyte solutions may be given according to the requirement with their serum levels guiding the therapy . Intravenous H2 blockers (ranitidine 50 mg 8 hourly) or sucralfate 1 gm 6 hourly through nasogastric tube should be given to prevent upper GI bleed due to strees induced gastric erosions. Ampicillin in the dosage of 1 gm 6 hourly should be given as a gut steriliser either intravenously or through the nasogastric tube. Use of neomycin for this purpose has more or less been abandoned due to its nephrotoxicity despite neomycin being poorly absorbed. Use of lactulose in FHF is controversial in contrast to its proven role in treating encephalopathy in patients with chronic liver disease. The reason for this is related to the pathophysiology of encephalopathy in FHF where ammonia is not a major factor. Instead, increased GABAergic neurotransimission (inhibitory in nature). and false neurotransmitters play important roles.[7] Nevertheless., some hepatologists do recommend lactulose in the treatment of FHF. Treatment modalities such as corticosteroids, lactobacilli, dopaminergic drugs, branched chain aminoacids are of no proven benefit. Fresh frozen plasma is recommended in patients with severely deranged coagulogram. Prophylactic use of FFP may result in fuild and electrocute disturbances, an increase in ICP. Use of prophylactic antibioics is not yet settled. Some workers have shown marginal benefit by the use of prophylactic antibiotics (selective parenteral and enteral antibiotic regimen -SPEAR) which however, did not result in increased survival.[8]

Specific therapy: Acetylcstenine iv (150 mg/kg over 15 min. 50 mg/kg over next 4 hours and 100 mg/kg over next 16 hours repeated every 16 hours till recovery) has been recommended in acetaminophen induced FHF. The mechanism is by providing glutathione which converts acetaminophen into inactive metabilites. The drug should be given preferably within fisrt 10-15 hours. It has also been shown to be of some benefit in FHF in general by maintaining hemodynamic stability.

Treatment of Complications:

Cerebral edema: Mannitol 20% in the dosage of 1 gm/kg i.v 6-8 hourly is the drug of choice.[6] If it is ineffective pentobarbital or thiopental iv may be required. Hyperventilation may also be tried. We use mannitol in all patients with grade III, IV coma prophylactically also. Use of mannitol has been shown to improve survival in patients with FHF. The plasma osmolarity should not increase >320 mosmol. Steroids have no role.

Infection: Ideally culture and sensitivity reports should guide the choice of antibiotics. In absence of that, broad spectrum antibiotics should be started. Aminoglycosides should be avoided in patients with liver disease because there are reports of increased incidence of nephrotoxicity. If pseudomonas infection is suspected, ceftazidime is a good choice. Some patients may require antifungal agents.

Bleeding: FFP should be transfused. The requirement may vary from 1-2 litres/day. For GI bleed intensive antiacid regimen should be employed i.e iv ranitidine 12.5 mg every hour and antacid drip 0.5 ml/min through the nasogastric tube.

Acute Renal Failure: In addition to conservative management of ARF, hemodialysis should be offered to these patients. A better alternative in the form of continuous venovenous hemaodialysis (CVVHD) has recently emerged which can be done at patients bedisde.

DIC: In presence of overt bleeding, FFP may be required. Use of heparin is contraindicated.

ARDS: Assisted ventilation is mandatory. At many centres, all patients with FHF are intubated routinely and those with hypoxia, deep coma and signs of cerebral edema are electively ventilated.

Liver Support:

Artificial Liver Support: Many devices have been used which include charcoal hemoperfusion[9], cross circulation through baboon or human, plasmapheresis etc. but none has been shown to be of any benefit. The problem is that the total amount of blood flow through these devices is miniscule compared to that through the liver in vivo. Recently hemofiltration through hollow fibres containing live hepatocytes obtained from cell cultures (Extracorporeal liver assist device) has been found to be useful in sustaining the patient for short periods of time.

Liver Transplantation: Orthotopic liver transplantation (OLT; grafting donor liver after removal of patient's own liver) has dramatically reduced the mortality in patients with FHF from 60-90% down to 20-30%. The major problem is selection of patients for OLT. The fact that upto 40% of patients may recover with medical treatment alone will make OLT necessary in as many patients and such patients will be left with lifelong immunosuppressive with all its attendant complications. This also suggests that the total cost of treating such patients will necessarily be very high. This therefore calls for highly sensitive and specific criteria for selection of a patient for OLT. Two such sets of criteria are mainly in use (table 4.),[10]

Auxiliary liver transplantation (transplanting reduced size donor liver keeping patients's own liver in place) is also being done with the hope that in the event of regeneration of the native liver, mere stoppage of immunosuppressants will get rid of the unwanted grafted liver.

Hepatocyte Transplantation: This is an exciting area in the treatment of FHF which has generated much interest. It theoretically offers the advantages of liver transplantation to sustain the patient for the period during which his/her own liver regenerates while avoiding the high cost, rejection problems and lifelong immunosuppression associated with OLT. Live hepatocytes are taken from cell culture lines and placed either into the spleen, intraperitoneally or lungs. Preliminary results show some benefit but the techique needs further refinement and more study before it can be used in patients.[11]

Address for Correspondence:
Prof. R.K. Tandon,
Department of Gastroenterology,
A.I.I.M.S., New Delhi 110 029.
Tel: 6594885, Fax: 6862663

Table 1. : Aetiology of Fulminant hepatic Failure

Viral Hepatitis
- Hepatitis A, - Hepatitis B, - Hepatitis E ,
- Hepatitis D
(coinfection with B)

Drugs
- Isoniazid, - Rifampicin, - Pyrazinamide
(with INH and Rifampicin), - Acetaminophen,
- Phenyton, - Sodium Valproate*, - Tetracycline*

Vascular
- Acute Budd Chiari Syndrome, - Shock

Metabolic
- Wilson's disease, - Galactosemia, - Tyrosinemia

Miscellaneous
- NonHodgkins Lymphoma,
- Acute fatty liver of pregnancy* , - Severe sepsis

Toxins
- Carbon Tetrachloride, - Phosphorus, - Aflatoxins

* cause microvesicular steatosis

Table 2: Grading of Hepatic Encephalopathy

Grade	Description
I	confusion, altered behaviour, altered sleep rhythm, drowsiness
II	delirium, asterixis, slow mentation, disorientation
III	stupor, transiently arousable, +asterixis
IV	coma, may respond to painful stimuli, asterixis absent, in deeper coma, no response to painful stimuli.

Table 3 - Checklist for daily monitoring

Clinical:

Hourly -
Pulse
Blood pressure
Respiratory rate
Temperature

2-4 Hourly -
Central venous pressure
Papillary size and reaction
Signs of cerebral edema
Throat secretions
Urine output

Daily -
Liver span
signs of sepsis

Investigations:

Twice a day -
Serum sodium
Serum potassium
Blood sugar
Blood urea
pH, arterial gases

Daily
Prothrombin time
Haemoglobin
Total leucocyte count
Serum creatinine
Serum bilirubin
GI Bleed
Grade of coma

Table 4 - Indications for liver transplantation

Prothrombin time > 100 Sec.
or
Any three of the following

- Age <10 or >40 years
- Drug induced or Non A non B hepatitis
- Encephalopathy after 7 days of jaundice
- Serum bilirubin >17.7 mg.dl
- Prothrombin time >50 Sec

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Nirasons

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HOECHST INDIA LTD.
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Note :- Anti-Cancer Drugs also available

Applications of Laser in Surgery

Nazir Ahmad Wani*; Omar Javeed Shah; Khursheed Alam Wani***; G.Q. Peer****; Syed Qawnain*****

JK Practitioner 1996: 3:4 220-221

Surgical laser is a device capable of generating electromagnetic energy in the ultraviolet, visible and infrated regions of the spectrum. The monochromatic, coherent light that is generated can be used efficiently for surgical applications. Taking advantage of the pure colour of the laser's emission, diseased tissue can be selectively treated by matching the absorption characteristics of the tissue to the wave length. The coherent property of the laser's emission permits the efficient utilization of the power that is generated and the coherence permits the precise focusing of the beam of the light.

The word LASER is an acronym for light application by stimulated emission of radiation. Lasers that currently have found the widest applications in medicine are those based on Carbon dioxide (co_2), argon, neodymium, Yttruim aluminium garnet (Nd. YAG), Krypton and various dyes. In General Surgery the Co_2 and contact Nd: YAG lasers are the main ones used.

Laser has two fundamental characteristics:

1. Production of identical photous by a stimulated emission process.
2. Amplification of this stimulated emission

A pump source is used to excite the active medium. As simply a form of electromagnetic energy that is capable of being absorbed by the atoms in the active medium. The pump source depending on the type of active medium being utilized can be a light electric current, a high voltage, ora radiant source such as another laser or intense light bulb. After the atoms of the active medium aborb the energy from the pump source, they spontaneously emit light in all directions. If a sufficient number of atoms are excited, the photous travelling along the axis of the cavity will colloid with each other excited atoms resulting in two photous of light travelling in phase with each other and in the same direction. These photous will colloid with other atoms thus multiplying the number of coherent photous. At each end of the active medium are mirrors (reflectors) that contain the photous of light within the cavity. One of the two mirrors is partially reflecting, thus permitting a fraction of the photous of light to escape out of the cavity. The energy passing out of the cavity is in the form of an intense beam of monochromatic, coherent laser light. The photous contained within the cavity continue to oscillate between the two mirrors rapidly amplifying the number of photous travelling along its axis. The first laser Maiman in 1960 used ruby as a medium. Now a wide range, of solids, gases liquids or vapours are available as lasing media. The latest laser technology developed is the diode laser. Diode lasers are electronic semiconducting devices that emit laser light as electrical current passes through them.

Clinical Applications:-

Liver:

Experimental studies have already shown the possibility of liver resection with Co_2 or Nd-YAG laser. There is significant reduction in operating time, decreased peri-operative blood loss but more extensive tissue necrosis was observed.

Recently Katkouda et al (1992)[3] has reported the first case of laparoscopic Hydatid cyst of liver resection. In this report a Nd-NAG laser was used.

Biliary:

Laparoscopic laser cholecystectomy has been proposed as an alternative to electrocautery (Hunter 1991)[4], Reddick and Olsen (1990)[5].

Common bile duct stones may be fragmented using the photoacoustic effect of laser beam. In this contact technique, the laser light induce a plasma, which when hot produces a shock wave which fractures the stone. (Gitomer and Jones 1987)[6].

Pancreas:

Pancreatic resections have been done using Nd-YAG laser both in experimental and clinical prac-

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**Prof. and Head Department of Surgery.*
***Lecturer Department of Surgery.*
****Assistant Professor Department of Surgery.*
*****Associate Professer Department of Surgery.*
******Senior Resident Department of Surgery*
Department of General Surgery Sher-i-Kashmir Institute of Medical Sciences, Soura, Srinagar, Kashmir.

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tices (Joffe et al 1985)[7]. A significant reduction in operating time and blood loss was observed.

Spleen:

(Disen et al 1980)[8] have described open technique for segmental or partial splenectomy.

Head and Neck:

Head and neck tumours have been excised by using Co_2 laser (Aroneff 1981)[9]. Excision of head and neck lymphangiomas have been done using Co_2 laser (White and Adkins 1986)[10].

Oesphagagastrointestinal:

Contact ND-YAG laser has been advocated as a useful instrument for viseral dissection (exp. stomach and intestine) during open procedures (Hire and Moore 1987)[11]. Endoscopic laser surgery has been used as a palliative therapy of gastric, intestinal malignancy. In various cancers of oesophagus, stomach, colon and rectum, laser has been used to relieve dysphagia, obstruction or to control bleeding.

Cutaneous lesions.

Argon laser is a useful instrument for the treatment of various cutaneous lesions e.g. Congenital port wine stains and other vascular lesions. However it produces very satisfactory results when applied to professional decorative tattoos.

Gynaecology:

The most common indication for laser surgery in gynaecology is the treatment of cervical intraepithelial Neoplasia. It is further used in condylomata acuminata and genital herpes infection. Laser has been widely used in treatment of infertility. Various causes and contributors of infertility such as endometriosis, tubul occlusions, scar tissue and adhesions can now be treated with the Co_2 laser. The laser can be used intra-abdominally for adhesiolysis, fimbroplasty, salpingostomy, tubul reanastamosis and cornual reimplantation.

Urology:

Preliminary work involving endoscopic and open application of the neodymum YAG laser to various malignant and benign urologic lesions has shown a high success rate which include its use in Bladder Cancer, transitional cell cancer of renal pelvis and ureter and urethral structures.

Open application of laser photoradiation has been used in penile carcinoma, condylomata acuminata and malignant melanoma of penis.

Upper Air way:

Co_2 Laser is specifically indicated for the treatment of laryngeal papilloma, vocal cord polyps, nodules, hemagioma and granuloma, Laryngeal and subglottic stenosis and laryngeal malignancies.

Opthalmology:

Laser beam therapy can be used to perform a variety of surgical procedures for example, iridotomies, trabeculoplasty, photomydriasis, capsulotomy and vitrectomy in the anterior segment or panretinal photocoagulation and focal therapy in the posterior segment.

Photo dynamic therapy and photodynamic imaging.

Photo dynamic therapy and imaging are two interesting applications of non-thermal lasers based on the selective retention of photosensitizes by malignant tissues and abnormal stroma. Absorption of light by the photosensitized produces either free cyto toxic radicals (tumour destruction) or fluorescence (tumour detection).

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Ischaemic Strokes:- An Update

Parvaiz A. Shah*; A. Nafee**

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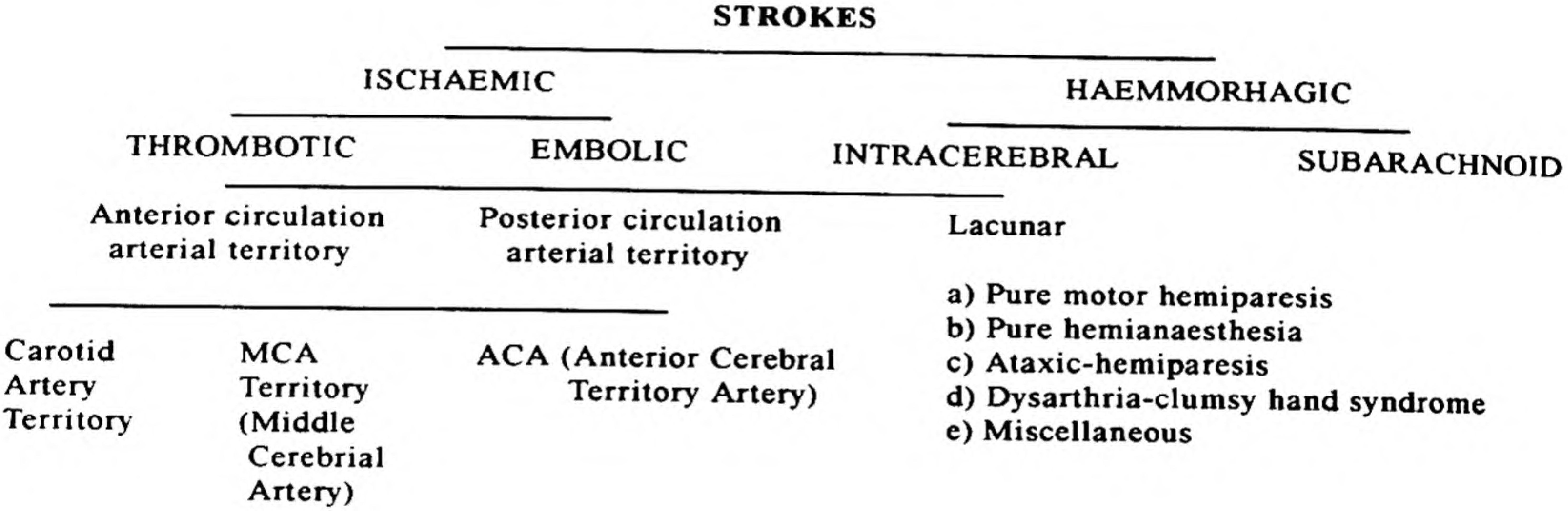
Stroke is the commonest cause of death after coronary artery disease and cancer. It is also the commonest cause of severe physical disability and around 25% of all strokes affect people under 65 years of age. In West, the annual incidence of stroke is two per 1000. The annual incidence of stroke in India varies from 57 per 100,000 in Vellore to 143 per 100,000 in Kashmir[1].

Transient Ischaemic attack (TIA) is defined as an acute, focal neurological deficit, resulting from vascular disease that resolves in less than twenty four hours. Majority of TIA's result from artery to artery embolism and more than 50% resolve in less than five minutes. TIA's are more common with thrombotic strokes than with cardio-embolic strokes. About 10% patients with TIA's develop stroke within one week.

Chances of developing stroke are more with crescendo and Vertebrobasilar TIA"s. Longterm risk for ischaemic stroke after TIA's is 5% per year[1].

Stroke is not a diagnosis. It is a description of symptom complex thought to have a vascular aetiology. It needs to be emphasised that stroke should be classified according to the anatomy of the lesion, its timing, aetiology and pathogenesis.

Classification of stroke


STROKES
ISCHAEMIC
HAEMMORHAGIC
THROMBOTIC
EMBOLIC
INTRACEREBRAL
SUBARACHNOID
Anterior circulation arterial territory
Posterior circulation arterial territory
Lacunar
a) Pure motor hemiparesis
b) Pure hemianaesthesia
c) Ataxic-hemiparesis
d) Dysarthria-clumsy hand syndrome
e) Miscellaneous
Carotid Artery Territory
MCA Territory (Middle Cerebral Artery)
ACA (Anterior Cerebral Territory Artery)

Anatomy

Carotid artery occlusion presents with contralateral hemiplegia, hemisensory loss, language dysfunction, homonymous hemianopia, gaze palsy contralateral to the side of lesion, horner's syndrome, central retinal artery occlusion or episodes of amaurosis fugax. Horner's syndrome classically occurs with carotid artery dissection which may be associated with ipsilateral IX, X, XI cranial nerve palsies. Internal carotid artery occlusion is invariably associated with increased flow across the superficial temporal artery and an orbital bruit.

Vertebrobasilar arterial territory occlusion results in visual hallucinations, field defects, cortical blindness, agitated delirium, cranial nerve palsies, gaze palsies, hemisensory defects, signs of cerebellar dysfunction and hemiparesis or tetraparesis. A wide range of other brainstem syndromes may follow ischaemia of localised area of brainstem.

The "Locked-in" syndrome results from infarction of pontine tegmentum. Here, the patient is fully conscious but the only possible motor activity is vertical movements of the eyes. On first glimpse, patient may appear to be unconscious.

**Consultant Neurologist*
***Professor Deptt. of Medicine*
Medical College Srinagar Kmr. 190010 (India)

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Lacunar Infarcts

These infarcts result from occlusion of small vessels of 100-400 um in diameter. They are commonly seen in patients with long standing hypertension and diabetes mellitus and tend to occur commonly in vertebrobasilar arterial territory.

Water Shed Infarcts

These infarcts account for 10% of all ischaemic strokes, and usually follow haemodynamic compromise. Watershed zones of the brain are located at following sites, a) Parieto-occipito-temporal junction (Borderzone between anterior, middle and posterior cerebral arteries). The parieto-occipito-temporal triangle is most commonly affected following a haemodynamic compromise. b) Fronto-parietal cortex corresponding to arm region. It represents junction between anterior and middle cerebral arteries. These patients present with bibrachial paresia (Man in barrel syndrome). c) Cerebellar infarction in the borderzone between superior cerebellar and anterior inferior cerebellar artery d) subcortical infarct in the borderzone between Heubner's artery (branch of anterior cerebral artery) and lenticulostriate branches of middle cerebral artery.

Timing and Pathogenesis

Majority of TIA's are embolic and usually arise from internal carotid artery which is followed by the heart. Haemodynamic infarcts, although uncommon, tend to occur in patients with significant internal carotid artery stenosis. Haemodyanamic infarcts occur following caridac arrest or prolonged hypotension.

Aetiology of Ischaemic Stroke

A) Embolic
  - i) Artery to artery embolism
  - ii) Cardioembolic
  - iii) Paradoxical emblism

B) Vascutitis
C) Vasospasm
D) Hyperviscosity syndrome
E) Miscellaneous Drugs like oral contraceptives, ergots, etc.

Risk Factors for Ischaemic stroke includes:-

a) Age
b) Hypertension
c) Cardiac disease
d) Diabetes mellitus
e) Smoking
f) Hyperlipidemia
g) Raised homocysteine levels
h) Raised fibrinogen levels
i) obesity

Differential diagnosis of an ischaemic stroke include epilepsy, intracranial space occupying lesion, migraine, primary demyelination, chronic subdural haematomia and intracranial vascular malformation.[2]

Partial motor seizures may mimic TIA's originating from carotid arterial territory. Focal sensory seizures need to be distinguished from hemisensory stroke.

Intracranial space occupying lesion may rarely present with so called tumour transient attacks. Vascular headache (migraine) may uncommonly present with neurodeficit. however, presence of aura symptoms favours diagnosis of migraine. Patients with migraine usually have positive symptoms in contrast to amaurosis fugax where patients usually complain of misty vision or blackness. Moreover, headache is an unusual symptom in TIA's or ischaemic stroke.

Neurological deficit resulting from primary demyelination is characterized by relapses and remissions and persistence of neurodeficit at least for twenty four hours. Occasionally, chronic subdural haematoma may mimic a TIA or an ischaemic stroke. Sometimes, chronic subdural haematoma may present with a picture of ataxic hemiparesis. However, a history of cranio-cerebral trauma and evidence of raised intracranial pressure corroborated by positive neuroimaging studies help to arrive at an appropriate diagnosis.

Investigations

a) Computerised Tomographic Scan (C.T. Scan)

By 8 hours after an infarction, about 20% of the cranial CT Scans (Plain) will be positive. It is unusual to have a negative CT scan 24 hours after an infarct. Moreover, CT also helps to differentiate an acute haemorrhagic stroke from an ischaemic stroke. Magnetic Resonance imaging is more sensitive in identifying parenchymal brain lesions than a cranial CT scan [3]

b) Lumber Puncture

Examination of cerebrospinal fluid per se, is not of much help when ischaemic stroke has resulted from atherothrombosis or cardiac embolism, However, cerebrospinal fluid analysis, is helpful in following situations: i) evaluation of a young patient with stroke ii) stroke thought to be secondary to central nervous system infection like syphilis, T.B.M etc. iii) Haemorrhagic transformation of an infarct with seepage of blood into the ventricular system.

c) Cerbral Angiography :- It includes:-
i) Four vessel conventional angiography ii) Digital subtraction angiography iii) Magnetic Resonance angiography.

Cerebral angiography is mandatory before contemplating surgery for an intracranial vascular lesion.

d) Non-invasive evaluation of the carotid arteries by i) Duplex scanning which is especially helpful in diagnosing carotid bifurcation disease ii) Transcranial doppler which provides an information about blood flow velocities and stenotic lesions in intracranial vessels.

e) Electoencephalography (EEG)

It is usually abnormal in cortical infarcts. EEG is charactistically normal in lacunar infarcts. It also helps in identifying non- conculsive status epilepticus in stroke patients.

f) Functional Neuroimaging

It includes positron emission tomography (PET) and single Photon emission tomography (SPECT). These techniques determine regional cerebral blood flow, cerebral blood volume and metabolic activities of the brain parenchyma. PET/SPECT become positive before CT scan or MRI. Functional neuroimaging also yields positive results in patients with TIA's where other neuroimaging techniques are noncontributory.[3]

In recent past, magnetic resonance spectroscopy (MRS) has been introduced in the evaluation of strokes. However, it is not yet widely available.

g) Every patient with cerebrovascular accident should have following baseline investigations like complete blood count, ESR, blood urea nitrogen, blood sugar and lipid profile done.

h) Echocardiography - It is indicated in following groups of patients:-

i) A patient with suspected cardiac lesion
ii) Young Patient with stroke if no apparent cause for stroke is discernible.
iii) Patient with multiple territory infarcts. Transoesophageal echocardiography is more accurate than routine 2-D echocardiography in detecting mural thrombi and valvular vegetations.

i) Holter monitoring in patients suspected to have cardio-embolic stroke subsequent to paroxysmal arrhythmias.

j) Miscellaneous:-

Following investigations should be considered especially in a young patient with cerebrovasular accident in whom aforementioned investigations have failed to arrive at a plausible diagnosis. These investigations should be done in selected patients only:-

- Serology for syphilis
- Collagen profile
- Protein C and protein S estimation
- Screening test for homocystinuria
- Muscle biopsy
- Brain/Meningeal biopsy
- HIV screen

Treatment

A) General Measures:

i) Supportive Care:- It includes maintenance of airway, vitals, care of bowel and bladder, input-output charting, back care and physiotherapy to prevent development of deep venous thrombosis, aspiration pneumonia and contractures.

ii) Patients with recurrent vomiting (usual feature of posterior circulation strokes), palatal palsy and mental obtundation should have nasogastric tube introduced.

iii) Intravenous fluids should be carefully administered in such patients as an injudicious use of intravenous fluids may lead to cerebral oedema. On an overage, daily input should preferably not exceed one litre in absence of fever or fluid loss.

iv) Overzealous lowering of blood pressure should be avoided during first 7-10 days after the ictus. Antihypertensive agents may be administered in following situations:-
   a) Patient with hypertensive encephalopathy
   b) Patient with left ventricular failure.

(v) Anticonvulsants like phenytoin 5 mg/Kg/day to be administered if patient presents with seizures. Dilantinization should be considered if patient presents in status epilepticus.

vi) Antioedema measures are to be instituted if there is evidence of raised intracranial pressure. Commonly employed osmotic agents are mannitol or glycerol. In an adult, mannitol is administered in a dosage of 0.25-1g/kg intravenously. Mannitol should not be used if plasma osmolality is more than 320 mosml/litre or if there is evidence of renal failure. Glycerol is administered in a dosage of 4 gm/kg in four divided doses.

B) Hyperacute therapy:

i) Heparin is administered intravenously in a dosage of 10,000 I.U. as a bolus followed by an infusion at the rate of 1000 I.U per hour International normalized ratio (INR) should be maintained at twice the control value. In cerebral embolism study group, all patients with symptoms of less than 48 hours duration showed satisfactory response to heparin therapy with no recurrence of symptoms or haemorrhagic transformation of infarct.

Chamorro et al (1995) advocated that early heparin administration should be safely considered in non-septic embolic strokes in following situations. a) Small infarct b) Alert patient. However, partial thromboplasim time should be strictly monitored in these cases and in no case should exceed three times the control value. Heparin administration should be followed by oral anticoagulation like Warfarin sodium (2 to 15 mg daily) [4]

ii) Thrombolytic therapy

It is not routinely used. Thrombolytoic agents used include streptokinase, urokinase and t-plasminogen activator (r-TPA). r-TPA is currently being tried in various controlled trials and is preferred to urokinase or streptokinase.

iii) Surgery

Carotid endarterectomy should be considered in young patients with symptoms of less than 24 hours duration with adequate collaterals as shown by cerebral angiography. The procedure carries the risk of cerebral oedema following hyper-perfusion and haemorrhagic transformation of the infarct.

C) Primary Prevention

It envisages control of modifiable risk factors like hypertension, artrial fibrillation and smoking.

D) Secondary Prevention

I) Pharmacologic Agents

Aspirin in a dosage varying form 30mg to 1300 daily has been found to reduce incidence of stroke by 25% .Aspirin is commonly used in daily dosage of 325 mg, Dipyridamole in a dosage of 50 to 100mg thrice daily has not been found to be superior to aspirin in stroke prevention.

Ticlopidine 500mg daily has been found to be beneficial in stroke prevention in patients who are unresponsive or intolerant to aspirin[5]

II) Surgery

Carotid endarterectomy in a patient with carotid stenosis has been a debatable issue. However, prophylactic carotid endarterectomy may be considered in an asymtomatic patient with carotid stenosis of >70%. NASCENT showed an 18% reduction in the incidence of stroke in surgically treated patients than those receiving only antiplatelets. However, CASA-NOVA (1991) study group did not find any difference between surgically or medically managed asymptomatic patients.[5]

E) New Frontiers in Stroke Management

I) Administration of magnesium sulphate, monoganglioside GM-I and ancrod, intravenously within first twenty four hours of presentation have yielded encouraging results in pilot trials. Magnesium sulphate after crossing blood brain barrier results in vasodilation by inducing prostacyclin release. Monoganglioside GM-I helps in neuronal repair and regrowth. Ancrod acts by decreasing fibrinogen levels. In diabetics, picotamide, an antiplatelet agent with antithromboxane activity, has been found to retard progression of early athero-sclerotic lesions.[5,6]

II) Cytoprotective agents

These include (a) nomodipine (calcium channel blocker), b) excitatory aminoacid antoagonists like lamotrigin, lubeluzole (pre-synaptic sodium channel blockers) and aptiganel hydrochloride, Ketamine dextromethorphan, selfotel, felbomate, eliprodil, and ifenprafil (all postsynaptic NMDA receptor blockers). In experimentatl trials, these agents have been found to reduce infarct size as well[5,6]

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Gastrointestinal Muscosal Protective Activity of Aluminum Ion-A

G. Nabi Mir*

JK Practitioner 1996; 3:4 226-229

Summary

Inorganic antacids have been extensively used in the treatment of peptic ulcer disease since the beginning of the present century. The effectiveness of antacids has been attributed to their ability to neutralize gastric acid secretion and inactivate pepsin. Recently clinical studies have shown that aluminum hydroxide treatment of peptic ulcer disease is comparable to other standard drug treatments (5). This review discusses studies that demonstrate that change in intragastric pH is not necessary for the antiulcer activity of $Al(OH)_3$ (1,2,3,4,5). In fact when $Al(OH)_3$ is deprived of its acid neutralizing capacity, the antiulcer activity increases 10 fold. The antiulcer activity of the substance may partially be due to increased in prostaglandin synthesis in the gastric mucosa and partially due to the mucosal defensive mechanisms.

Where is the mucosal protective activity coming from? If acid neutralizing capacity of aluminum hydroxide is not the activity, then some other property of aluminum ion is responsible for the change. We decided to investigate the activity and the mechanism systematically. Thus began a 12 year journey into mysteries of aluminum ion. The journey began at Rhone Poulenc-Rorer and moved to Ayerst Laboratories, Princeton, New Jersey, USA in 1983, where I was appointed as the Associate Director of Research.

Our investigation of animal pharmacology of aluminum hydroxide had began in early 1970's in response to internal clinical studies conducted by Rhone-Poulenc-Rorer on its aluminum hydroxide antacid Maalox, which had demonstrated significant duodenal ulcer healing property. In the latter part of the 1970's published clinical reports also started appearing(6) describing ulcer healing properties of aluminum hydroxide both in liquid and tablet forms and at doses much lower than the generally prescribed acid neutraluzing doses of aluminum hydroxide. In our investigation, we decided to study thoroughly preclinical mucosal protective and antiulcer profile of aluminum hydroxide. Before I go any further, let me define mucosal protective mechanism. "Mucosal protective activity is a defensive property of the GI tract and is differentiated from gastric antisecretory acitivity by the ability of an agent to protect stomachs from ulcer formation by activating the defensive mechanism, such as increase in prostaglandin synthesis, increase gastric mucos secretion, increase in DNA synthesis, increase in gastric mucosal sulfhydryl, e.g. reduced glutathione, increase in gastric mucosal blood flow etc. and thus strengthening the gastric mucosal barrier at doses which do not inhibit gastric acid secretion. Prostaglandins fall in to this category. Sucralfate is an mucosal protective antiulcer agent which does not inhibit gastric acid secretion, but stimulates gastric prostaglandin syntheses. Gastric acid inhibitory antiulcer agents such as H_2-receptor antagonists are not mucosal protective agents.

Gastrointestinal Mucosal Protective and Antiulcer Profile of Aluminum Hydroxide.

Male Sprague-Dawley rats, at 120-150 g and fasted for 24 hours were used. Various gastric, duodenal and intestinal ulcer models were absolute ethanol or acidified aspirin induced-gastric mucosal damage, indomethacin, stress or 18 hour pylorus-ligation induced-gastric ulcers-, cysteamine induced-duodenal ulcers-and indomethacin induced-intestinal ulcers. All models indicated are well established methods for investigating antiulcer activity of any new or known chemical entity. The antiulcer profile of aluminum hydroxide as compared with sucralfate and ranitidine is shown in Table 1. As the Table 1(3) indicates, aluminum hydroxide overall has a better antiulcer profile than ranitidine or sucralfate. Aluminum hyroxide is normally used as an antacid at pH 9 and above. Our first step was to gradually deprive aluminum hydroxide of its acid neutralizing capacity and test the partially or completely neutralized substance in the ethanol induced gastric mucosal damage model in the rat. One ml of liquid aluminum hydroxide (216mg/kg w/w of aluminum hydroxide) was neutralized in increments to a lower pH till the gel turned into transparent liquid and at each stage of lower pH, gel was tested in our ethanol mucosal injury model. Generally the gel

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*BSc. M.Pharm. MBA. Ph.D.
President & CEO
Drug Research Laboratories, Inc.
P.O. Box 394
Buckingham PA 18912, USA

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turned completely into liquid between pH 3.3 and 2.5 and Table 2 shows the results.

The result unequivocally demonstrated that as aluminum hydroxide is deprived of its acid neutralizing capacity and is turned into a free floating ion (pH 2.5), its gastrointestinal mucosal protective properties are much better than aluminum hydroxide and further increase in the neutralization (pH 1.0) does not increase the activity. The product at its maximum pharmacological acitivity has optimal pH of 2.5. A question still remained., how does this ion actually behave physically in the solution and how good is its total antiulcer profile in relation to aluminum hydroxide? Meanwhile, we named our new product of aluminum hydroxide pH 2.5 as Activity Aluminum Complex, (AAC) and filed an applicated for US patent(2).

Physico-Chemical Properties of Activated Aluminum Complex

Neutralization of aluminum hydroxide gel by hydrochloric acid was studied (7) by NMR under conditions of both equilibrium and kineic control. The NMR studies demonstrated formation of aluminum ion surrounded in a physical bond by 4 to 6 water molecules and in crystalline form its structure was octahedral. These cations were named as hexaaquoaluminum cations $[AL(H_2O)6^{3+}]$. The solution near pH 2.5 has acid dissociation equilibria shown below:

$$Al(OH)3 + H^+ \rightleftharpoons AL(H_2O)_4 (OH)_2^+$$

$$AL(H_2O)_4 (OH)_2 + H^+ \rightleftharpoons AL(H_2O)_5 (OH)^{2+}$$

$$AL(H_2O)_5 (OH)_2 + H^+ \rightleftharpoons AL(H_2O)_6^{+3}$$

The maximum concentration of hexaaquoaluminum cations was observed at pH 2.5 and decreased as the pH was lowered further. The pH profile for the appearance of hexaaquoalumimum cations corresponds well with mucosal protective activity of acidified aluminum hydroxide, where maximum mucosal protective activity was observed at pH 2.5. It was earlier reported (4) that the octahedral crystalline structure of aluminum hydroxide coupled with a surface charge of the molecule, was a major requirement for the expression of mucosal protective activity. The hexaaquoaluminum cations possess both of the above requirements: an octahedral shape and a positive surface charge. Thus, the increased concentration of hexaaquoaluminum cation on acidification of an antacid suspension may explain the enhanced mucosal protective property observed with acidified aluminum containing antacids.

Gastrointestinal Mucosal Protective and Antiulcer profile of Activated Aluminum Complex (AAC).

When a mucosal protective and antiulcer profile similar to aluminum hydroxide (Table 1) was prepared for AAC, the results as shown in Table 3 (1,2,4,5) are quite dramatic.

AAC demonstrated activity at all levels of the rat gastrointestinal tract, i.e, stomach, duodenum, and small intestine. It was active in three mucosal cytotoxic models, Ethonal and acidified aspirin induced gastric lesions an indomethacin induced intestinal lesions. It worded against NSAID induced gastric damage, against a gastric ulcer induced by hypersecretion of gastric acid by 18 hour pylorus-ligation, and against restrain-cold stress induced ulcers. AAC protected against duodenal ulcers induced by cysteamine. Besides aluminum hydroxide, AAC was the only agent active across the board in every ulcer model. Futhermore, it was shown (unpublished observation) that AAC has rapid onset of action (1-3 minutes) and long duration of action (10 hours). AAC does not effect gastric acid secretion and has no toxicity.

Concluding Remarks

The major finding of this investigation was the demonstration that when $AL(OH)_3$ was gradually deprived of its acid neutralizing capacity from an original value of pH 9.1 to pH 2.5, the gastric mocosal activity also increased gradually, until the activity was 10 times greater than the original value. This enhanced activity of AAC is due to the formation of hexaaquoaluminium cation, which causes PGE_2 release (unpublished observation) in the gastric mucosa suggesting that prostanoid may partially be involved in the mucosal protective activity. Indomethacin (unpublished Observation), which completely abolished the PG biosynthesis in the gastric mucosa, was not totally able to abolish the mucosal protective of AAC.

As has been demonstrated recently about the involvement of Helicobacter pylori and cytotoxicity independent of gastric acid secretion in the peptic ulcer disease,. the dictum of Schwartz in 1910. "without acid no ulcer," may not be true any more, and the assumption that various gastrointestinal diseases are characterized mainly by different acid secretion rates is not valid any longer.

Because of mucosal protective effects of AAC throughout the gastrointestinal tract, i.e, stomach,

Table 1: Effect of Aluminum Hydroxide, Rantitidine and Sucralfate on various Gastrointestinal Ulcer Models in the Rat.

		ED 50 (mg/kg, po)	
	Ranitidine	Sucralfate	Aluminum Hydroxide
Mucosal Cytotoxic ulcers			
Ethonal	NE	60	453
Acidified Aspirin	NE	58	689
Gastric Ulcers			
Indomethacin	6	148	239
Stress	4	194	382
18h Pylorus-ligation	20	64	620
Duodenal Ulcers			
Cysteamine	1	507	243
Intestinal Ulcers			
Indomethacin	NE	NE	432

NE= not effective

Table 2: Effect of Aluminum Hydroxide (216 mg/kg,w/w) on Ethanol-Induced Gastric Mucosal Damage in the Rat.

Treatment	Percent Inhibition of Gastric Mucosal Damage
Aluminum hydroxide, pH 9.1	44
Aluminum hydroxide, pH 4.4	68
Aluminum hydroxide, pH 3.3	86[a]
Aluminum hydroxide, pH 2.5	96[a]
Aluminum hydroxide, pH 1.0	73[b]

[a] $p<0.01$ vs Aluminum hydroxide, pH 9.1

[b] $p<0.05$ vs Aluminum hydroxide, pH 9.1

Table 3: Effect of Aluminum Hydroxide, AAC, Rantitidine and Sucralfate on Various Gastrointestinal Ulcer Models in the Rat.

		ED 50 (mg/kg, po)		
	Ranitidine	Sucralfate	$Al(OH)_3$	AAC
Mucosal Cytotoxic				
Ethonal	NE	60	453	58
Acidified Aspirin	NE	58	689	61
Gastric Ulcers				
Indomethacin	6	148	239	45
Stress	4	194	382	466
18h Pylorus-ligation	20	64	620	63
Duodenal Ulcers				
Cysteamine	1	507	243	69
Intestinal Ulcers				
Indomethacin	NE	NE	432	346

H=hour

NE= not effective

duodenum, and intestine, its therapeutic uses are being investigated in different forms of gastritis, stress induced gastric bleeding, and inflammatory bowl disease.

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"Tobacco and the Integument"

G.H. Hajini*; Iffat Hassan;
Parvaiz A. Shah***; Abdul Ahad******

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King James of England described smoking as a custom loathsome to the eye, hateful to the nose, harmful to the brain and dangerous to the lung". Cigarette smoking is detrimental to health and the skin is not immune to the adverse effects of tobacco smoke.

Cigarette smoke is a heterogeneous aerosol produced by combustion of the tobacco leaf which comprises of a gas phase in which particulate matter is dispersed. About 95% of the total weight of mainstream smoke exists in the gas phase out of which 85% comprises of nitrogen, oxygen and carbon dioxide. About 4000 substances have been isolated from cigarette smoke. Tar, armoatic hydrocarbons, nicotine, phenol, benzypyrene, trace metals, indole, catechol and carbazole are present in particulate phate whereas carbon monoxide, acetaldehyde, formaldehyde, ammonia, nitrosamines and vinyl chloride exist in the gas phase.

The addictive effects of smoking are primarily due to nicotine, polynuclear aromatic hydrocarbons, aromatic amines and nitrosamines are the major carcinogens in cigarette smoke, catechol, cresol and phyenol act as co-carcinogens. Cigarette smoking is the single most preventable cause of morbidity in the United States and each year is responsible for more than 400,000 deaths. Smoking accounts for about 3 million deaths per year all over the world with 2/3rds of the deaths occuring in the developing world (1-3). The death toll is expected to rise to 10 million per year by 2020, with 70% of these occuring in the developing world. In India, approximately 800,000 deaths are attributed annually to tobacco use. Large population studies have shown a strong association between cigarette smoking and several diseases. The incidence of coronary artery disease, strokes, peripheral vascular disease, bronchogenic carcinoma and various other malignancies is higher in smokers than in the general population. In India, nearly 1/3rd of cancers are attributed to tobacco use and the mortality rate in smokers is nearly 1.4 to 1.6 times higher than in non-users.

The cutaneous manifestations of smoking are nonetheless rare and occasionally may be associated with significant morbidity. However, a knowledge of the cutaneous effects of smoking may provide some patients with an effective motivation to stop smoking.

I. Wrinkles

Smoking predisposes to premature aging and

**Professor and Head,* ***Registrar,*
Department of Dermatology
*** *Neurophysician*
**** *Prof & Head,*
Department of E.N.T.
Government Medical College Srinagar Kmr. 190010

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facial wrinkling. Cigarette skin is characterized by pale grey colour with extensive wrinkling. Facial wrinkling has been found to increase with the number of pack-years smoked (4). Models criteria (5,6) for smolder's face are as follows:

a) Prominent wrinkles b) Gauntiness of facial features with prominence of underlying bony contours c) Atrophic appearance of the skin d) Plethoric facies.

The exact pathogenesis of smoker's face has not been fully elucidated. However, following observation have been made in this context (7-10);

i) Elastin from skin in smokers has been found to be thicker as compared to controls. ii) Increased neutrophil elastase activity iii) Blocking of cross linkage of elastin iv) Chronic ischaemia of the dermis leading to decreased collagen synthesis v) Decreased vitamin A levels in skin which is associated with poor protection against free radicals. vi) Increased hyperestrogenic state resulting from increased hydroxylation of estradiol. vii) Genetic predisposition.

II. Wound Healing

Mosley and Finseth (II) Postulated that smoking retards wound healing. Their view was upheld by Laurence et al (12). The average scar width in midline incisions has been found to be 7.4 mm in smokers as compared to 2.7 mm in non smokers (13). The complications associated with skin grafting have been found to be severalfold higher in smokers than in non smokers (14). The detrimental effects of smoking have been attributed to following factors (15-18).

a) Nicotine induced vasoconstriction. b) Peripheral vasoconstriction resulting from smoking induced sympathetic stimulation. c) Enhanced formation of chalones which retard epitheliazation. d) Decreased tissue oxygenation. e) Increased carboxyhaemoglobin levels which decrease oxygen carrying capacity of blood. f) Increased platelet aggregation and decreased prostacyclin synthesis. g) Increased blood viscosity. h) Reduced collagen deposition.

III. Squamous Cell Carcinoma

Keratocanthomas have been found to be more common in smokers than non smokers. Smoking is one of the predisposing risk factors for the development of squamous cell carcinoma (19). However, no association has been found between smoking and basal cell carcinoma.

IV. Anogenital Cancer

The risk of developing penile cancers is more in smokers than non smokers(20). More than 50% women with anal cancers have been found to be smokers(21). The risk of developing anal cancers in smokers versus non smokers has been found to be to the tune of 7.7. Carcinoma of the vulva has been found to be significantly higher in smokers than non smokers(22). The presumed aetiology of developing squamous cell carcinoma among smokers include i)Immunosuppressive effects of smoking. ii) Direct carcinogenic effects of cigarette smoke. iii) Adverse effects of nicotine on various body tissues.

V. Lip Cancer

The prominent risk factors for carcinoma of lip include solar radiation and tobacco exposure (23). 80% of lip cancer patients have been found to be smokers(24).

VI. Oral Cancer

There is a strong association between oral cancer and smoking Alcoholism in combination with smoking enhances the risk significantly. All modes of tobacco abuse including reverse smoking are well know risk factors for oral cancer.(25).

VII. Leukoplakia

About 70-90% patients with leukoplakia have been found to be smokers. Leukoplakia has been found to be six times common in smokers than non-smokers. Malignant transformation of leukoplakia has been reported in upto 10% of cases (25-27)

VIII. Leukokeratosis Nicotini Palati

Leukokeratosis Nicotini Palati is an oral lesion seen exclusively is smokers. The lesion is characterized by keratosis of the hard palate with multiple umbilicated erythematous papules. The lesion is commoner in patients who indulge in reverse smoking and pipe smoking than in cigarette (28). The lesion results from the tars and thermal effects of tobacco smoke and resolves within two weeks of cessation of smoking. Leukokeratosis nicotini palatic resulting from reverse smoking may progress to oral cancer(25).

IX. Leukokeratosis Nicotine Glossi

leukokeratosis nicotine glossi (smoker's tongue) is characterised by leukoplakia with hemispherical depressions involving anterior two-thirds of the dorsum of the tongue (29). The lesion is seen is pipe smokers and has no malignant potential. The aetiopathogenesis is presumed to be the same as in leukokeratosis nicotini palati.

X. Acute Necrotizing Ulcerative Gingivitis

Actue necrotizing ulcerative gingivitis (Trench mouth) results from bacterial infection which is characterised by a punched out ulceration of the interdental papillae, pain, bleeding, fetid mouth and lymphadenopathy and occurs exclusively in smokers (25,30).

XI. Miscllaneous Oral Lesions

These include:-

a) Oral veruccous carcinoma b) Black hairy tongue c) Oral warty Dykeratoma d) Oral melanosis.

XII. Psoriasis

The association between smoking and psoriasis has been a subject of controversy. Braathen et al found that 60% of patients with psoriasis were smokers.

XIII. Palmoplantar Pustulosis

O' Doherty and Macintyre (31) reported that 80% of patients with palmoplantar pustulosis were smokers. They found that the relative risk of palmoplantar pustulosis in smokers was 7.2. The pathogeneis of palmoplantar pustulosis has not been fully elucidated. However, smoking may predispose to palmoplantar pustulosis by increasing peripheral neutrophil count and inducing various morphological changes in neutrophils(32).

XIV. Melanoma

Smoking is not a risk factor for melanoma. However, smokers with melanoma have a worse prognosis as compared to non-smokers. Smokers with melanoma differ from non-smokers with melanoma in the following respects:-

a) Smokers are more likely to have metastasis at initial presentation b) are more likely to have visceral metastasis c) are more prone to have metastasis within first two years after diagnosis.

The mechanism by which smoking worsens prognosis in such patients is probably related to the adverse effects of smoking on the immune system (33).

Address for Correspondence:
Dr. G.H Hajini
Prof. and Head
Department of Dermatology,
Govt. Medical College Srinagar Kmr.

XV. Miscellaneous

Following dermatologic conditions have been found to be more common/increasingly severe among smokers:-

a) Infectious eczematoid dermatis b) atopic dermatitis c) Buerger's disease d) Periductal mastitis e) Yellow brown discoloration of finger nails (Nicotine sign) f) Cervical human papilloma virus infection g) Genital condylomas in females.

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The Pattern of child Psychiatric disorders in Kashmir

Mushtaq A. Margoob*

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***Abstract:** Sex, age and diagnostic chararcteristics of a consecutive series of first contact children, seen over a period of one year at the sole psychiatric centre of Kashmir Valley, was analysed. Most of the cases (excluding epilepsy) had dissociative disorders (22.69%). The next largest number was that of mentally retarded (17.64%). 8.4% suffered from schizophrenia, 4.2% from neurotic disorders other than dissociative disorders. A significant finding of the Study is that 2.52% cases had post traumatic stress disorder.*

***Keywords:** Pattern, psychiatric disorders, children*

Introduction:

Child psychiatry is still comparatively a neglected field in the developing countries. Children form 40% of the total population in developing countries, where as they constitute only 25% of the population in the developed countries[1]. The recognition of psychiatric problems in children by adults is important, as it is they who determine whether and where consultation treatment will be sought.

In view of the paucity rather absence of any reported study in this field from the State of Jammu & Kashmir, the present exercise was undertaken to report on the age, sex, diagnostic characteristics and domicile of all children utilising the consultation/treatment services at the sole psychiatric teaching-cum-treatment centre of the Kashmir Valley over a one year period during the year 1995.

Material and Method:

The Study was conducted at the Hospital for Psychiatric Diseases, Srinagar, Kashmir, which is the sole psychiatric teaching-cum-treatment institution in the Kashmir Valley catering to the needs of the people from the Valley and adjoining areas of the Jammu province and the Ladakh region.

All cases of psychiatric problems met with in children registered during the year 1995 were included in the Study. The diagnosis was obtained from individual case record files. The diagnoses were based on WHO's ICD-10 Classification and reflected the opinion of the consultant or Senior Resident.

Results:

The diagnositc breakdown of the sample, as described in the Table, reveals that the maximum number of children (excluding 43 cases of epilepsy) suffered from dissociative disorders (27). The next largest number was that of mentally retarded (21). 10 cases suffered from schizophrenia, 5 from neurotic disorders other than dissociative disorders and 2 cases suffered from depression.

Besides three cases of post traumatic stress disorder, which is a significant finding of the Study, only 2 cases of conduct disorder and 1 case of hyperkinetic disorder reported for consultation during the Study period.

Discussion:

Even though the neurotic and conduct disorders constitute the commonest psychiatric problems in children[2], only a selected section of these cases seek expert psychiatric help. Very often a large number of such cases in our setting are handled by traditional healers and general practitioners. It is clearly reflected in this Study that the majority of the children attending the Hospital Outpatients Department with various psychiatric symptoms belong to the neuropsychiatric disorder group, which constitutes 57.15% (68) of the total sample. It includes 36.13% (43) cases of epileptic disorder; 17.64% (21) of mental retardation, and 3.36% (4) cases of hyperkinetic and conduct disorders. A preponderance of seizure disorder cases in the sample is in keeping with the findings of an earlier diagnostic breakdown study by the author and associates[3].

Epileptic disorders constituted 16.43% of the

**Consultant Department of Psychiatry*
Government Medical College, Srinagar, India

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total of that study sample. One reason for such a high percentage of epileptics in the Psychiatric Outpatients Department population, as already reported could be that there has not been a separate Department of Neurology in the main Hospital of the Government Medical College, Srinagar, since its inception in 1969. Although the Department of Neurology at the Institute of Medical Science, Soura, has been functioning for more than a decade now, a significant number of epileptic patients prefer to visit the Psychiatric Diseases Hospital, where they are provided with free medication on a regular two-weekly basis, which may be an additional incentive for the poor patients of the Valley. Moreover, when the data about psychiatric morbidity in the community from other places is considered, epilepsy stands out conspicuously there also as a major group amongst these disorders. This is borne out by a recent community based Study from Kerala[4], which reported epilepsy to be the largest group (32.92%) amongst five priority psychiatric disorders, including mental retardation, convulsive disorders, schizophrenia affective disorders and organic psychosis. Epilepsy has been included in the list of psychiatric priorities in the study on the basis of the argument, that any debate about the respective roles of Neurology and Psychiatry at community level is irrelevant, because epilepsy should be selected for active intervention, as it usually affects individuals at a young age, leading to a wide range of physical and psychological impairment and social disability, and effective relatively cheap interventions are available. Anyway, if epilepsy and mental retardation (which is primarily mental deficiency rather than mental disorder) are excluded from the sample of the present Study, the dissociative disorders, which constitute 22.69% of the total cases, will form 49% of the remaining sample. This compares well with the finding of the Collaborative Study of the Indian Council of Medical Research on Psychiatric Disorders of Children[5], in which 23% of cases were diagnosed as having hysterical neurosis.

Another interesting finding of this Study is that, where as 51.26% (61) cases of the total sample belonged to rural domicile, only 29.63% (98) cases of dissociative disorders belonged to rural areas. This paradoxical finding of 70.37% of cases from urban areas, when considered in the light of documented reports that a higher incidence of hysterical disorders appears among lower socio-economic groups, rural population and persons with little education, deserves some explanation here. Sophistication of parents of children residing in urban areas, awareness about the existence of treatment facilities, greater facilities of conveyance in urban areas as compared to rural areas alone can't explain it totally. What seems more likely is that the majority of neurotic problems in general and dissociative (hysterical) disorders in particular are mostly handled by faith healers in rural areas not only due to ignorance of the rural population regarding the availability of psychiatric treatment facilities for such problems, but also due to prevailing belief amongst rural people in ghosts and supernatural powers in the causation of hysterical disorders in particular. The sex ratio of 1 boy:3 girls is in keeping with the reported finding of hysterical disorders being two to five times more common in female than male adults[6]. The majority (68.06%) of the total sample and 81.48% of the dissociative disorders belonged to the age group of 12-16 years, which compares well with the ICMR multi-centred Study which reported 55% of dissociative disorders in the age group of 12-16 years olds[5].

The relative absence of common behavioral disorders of childhood like conduct disorders and hyperkinetic disorders in our sample possibly reflects an inability in the general population to recognise deviance among children, as well as the comparative stability of the family in our setting[7]. This pattern of a very small number of behavioural disorders compares well with other reports[8,9]. Another reason for the smaller number of such cases in our sample could possibly also be due to the fact that at our place, as well as at most of the other places, where no separate child psychiatric or neuropsychiatric services are available, children with only gross behavioural abnormalities get referred and entertained in the general psychiatric Outpatients Department as "Young adults", and not as "Children" in their own right. This view gets strengthened when considered in the light of the results of a recent Study conducted at the Child Psychiatry unit of the central Institute of Psychiatry, Ranchi Psychiatric clinic, in which behavioural disorders constituted 18.47% of the total children attending the clinic in contrast to only 3.36% of the total children in our Study[7].

As reported earlier[10], an increasing number of post traumatic stress disorders (PTSD) in our psychiatric population is a recent phenomenon, and is a significant finding in the present Study also. PTSD arises as a protracted response to a stressful event or situation of an exceptionally threatening or catastrophic nature which is likely to cause pervasive distress in almost anyone. Out of 3 cases of PTSD in children registered in the Outpatients Department, one boy belonged to the age group of 5-11

Pattern of diagnostic categories among the two sexes and three age groups with their domicile.

	TOTAL	SEX		AGE			DOMICILE	
		Male	female	0-5	6-11	12-16		
DIAGNOSIS	%(No)	%(No)	%(No)	%(No)	%(No)	%(No)	Rural	Urban
	100 (119)	45.38 (54)	54.62 (65)	3.36 (4)	28.58 (34)	68.06 (81)	51.26 (61)	48.74 (58)
Psychoactive substance use disorder	0.84 (1)	100 (1)	-	-	-	100 (1)	-	100 (1)
Schizophrenia	8.4 (10)	40 (4)	60 (6)	-	20 (2)	80 (8)	60 (6)	40 (4)
Mood disorders -depression	1.68 (2)	100 (2)	-	-	-	100 (2)	100 (2)	-
Neurotic disorders other than dissociative disorders	4.2 (5)	20 (1)	80 (40)	-	20 (1)	80 (4)	20 (1)	80 (4)
Post traumatic stress disorder	2.52 (3)	33.33 (1)	66.67 (2)	-	33.33 (1)	66.67 (2)	66.67 (2)	33.33 (1)
Dissociative disorder	22.69 (27)	25.93 (7)	74.07 (20)	-	18.48 (5)	81.48 (22)	29.63 (8)	70.37 (19)
Hyperkinetic disorder	0.84 (1)	100 (1)	-	100 (1)	-	-	100 (1)	-
Conduct disorder	2.52 (3)	66.67 (2)	33.33 (1)	-	33.33 (1)	66.67 (2)	-	100 (3)
Sleep disorder (night terror)	0.84 (1)	-	100 (1)	-	100 (1)	-	-	100 (1)
Nocturnal Eneurisis	1.68 (2)	50 (1)	50 (1)	-	-	100 (2)	50 (1)	50 (1)
Mental retardation	17.64 (21)	38.1 (8)	61.9 (13)	14.28 (3)	38.1 (8)	47.62 (10)	66.67 (14)	33.33 (7)
Seizure disorders	36.13 (43)	62.79 (27)	37.21 (16)	-	34.88 (15)	65.12 (28)	58.14 (25)	42.86 (18)

years, and the two girls belonged to the age group of 12-16 years. This disorder needs due treatment consideration, as it can sometimes lead to enduring personality changes in a person's pattern of perceiving relating to or thinking about the environment and self.

Although, in contrast to an earlier finding[11] of 7.4% drug abusers below the age of 16 years, the drug abuse related problems constitute only 0.84% of cases in this Study, but in view of the age old peculiar patterns of drug use in Kashmir[12] and recent reports of smuggling of huge quantities of heroin across the Jammu border[13], and the use of cough syrups and other opium derivatives, by the younger population in the State[14], can have grave implications if corrective steps are not taken in time.

Therefore, clinicians and health care planners need to focus attention on factors that best predict child psychiatric service needs and utilisations. it would aid the achievement of a positive state of the physical, mental and social health of our progeny.

Correspondence:
Dr M.A. Margoob, P.O. Box 701,
GPO Srinagar 190 001.

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Tumor Pattern in Undescended Testis

Nasser D. Choudry*; A.R. Khan**

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***Abstract:** Out of 112 cases of testicular neoplasms recorded in last 12 years (1984-1995), 14 cases occurred in undescended testis. Five cases had bilateral tumors in bilaterally undescended testis. All the tumors were of Germ cell origin. They included seminoma 11 cases and one case each of Yolk Sac tumor, Teratoma and Yolk Sac Tumor combined with seminoma. Right testis was involved in 6 cases left in 3 cases and both in 5 cases. patients presented at age varying from 4 years to 70 years with mean age 31 years.*

***Key Words:** Undescended testis, tumors, bilateral, germ cell.*

Introduction:

Undescended testis is well established risk factor for the development of testicular neoplasm (1-5) Males with history of Cryptorchidism, have 40-50 times increased risk of developing tumor in that testis than normal scrotal testis (2). However, the magnitude of this risk is a point of considerable disagreement (5). Different studies on the subject available in literature are based on hospital population (case control) and so cannot be applied to general population (3). Successful early Orchidectomy does not lessen the risk of testis becoming neoplastic (6). Undescended testis occurs more commonly on right side than left and consequently accounts for the preponderance of tumors on right side (7).

Material and Method:

14 cases of tumors arising from undescended testis were recorded in the Department of pathology, Government Medical College, Srinagar during last 12 years (1984-95). Their clinical summary was obtained from the record. Histological slides were reviewed and wherever necessary fresh sections were cut from paraffin embedded blocks. Special stains used included periodic acid Schiff's, Alcain blue, Van-Gieson and reticuline staining. Hematoxilyne and Eosine was the routine stain employed.

Observation:

For clinical summary refer table (1). Material included Archidectomy specimen in 13 cases and cervical lymphnode in one case (14) who had widespread metastasis including pulmonary deposits and was clinically diagnosed as non-Hodgkins lymphoma.

Pathological Findings:

Seminoma: Largest tumor measured 200x160x50 mm. No normal testicular tissue was identified in this tumor. Smallest tumor was 90x40x30 mm with small compressed remnant of normal testicular tissue. Cord was infiltrated in three cases. Para-aortic node enlargement was seen (radiologically) in 4 cases at the time of diganosis. Cut surfaces were grey white homogenous lobulated with haemorrhagic areas in three cases and necrotic areas in 4 cases. Microscopy revealed typical morphology of seminoma with uniformally large cells arranged in sheets and nests, separated with fibrovascular stroma with varying amount of lymphocytic infiltrate. Lymphod follicles with germinal center was seen in one and epitheloid cell granuloma in 3 cases.

Yolk Sac Tumor:

Only case of Yolk Sac tumor occurred in 4 years old child who presented with abdominal mass, which on laprotomy appeared a retroperitonial tumor adherent to part of large gut. On gross it appeared a well circumscribed, encapsulated mass 100 mm in diameter, soft shinny. Cut surface revealed soft fleshy surface with areas of haemorrhage. Tumor seemed divided in two halves by thick fibrous bands. Microscopy Showed a typical Yolk Sac tumor with inter-communicating spaces of varying sizes lined by low columnar cells with loose fibrovascular intervening stroma and Schillar Dual bodies.

Retrospectively both testicles of this child were found absent

Diagnosis: Bilateral yolk sac tumor.

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*Demonstrator
**Professor of pathology,
Govt. Medical College.
Srinagar, Kashmir-India

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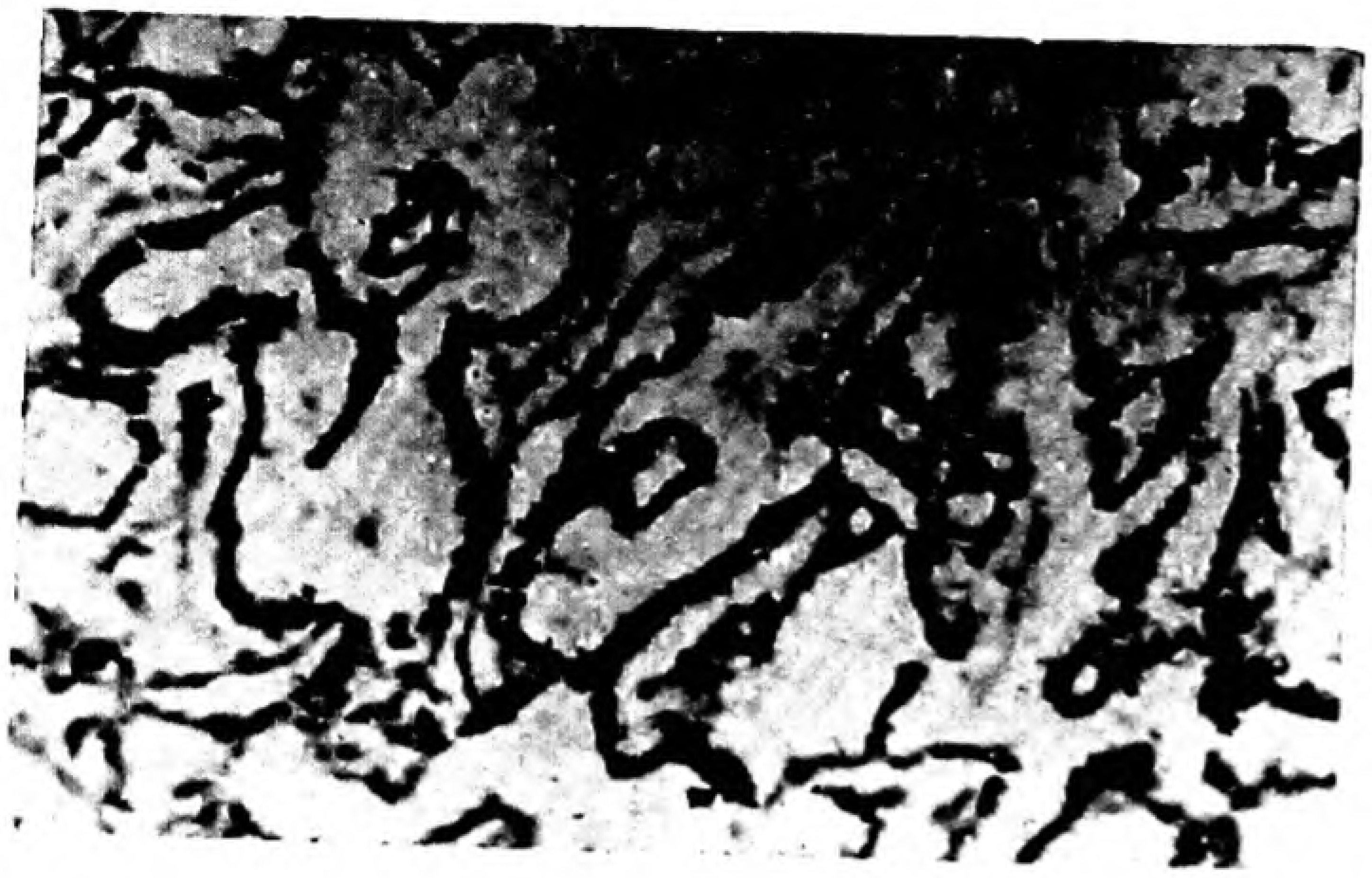

Fig. I Yolk Sac Tumor

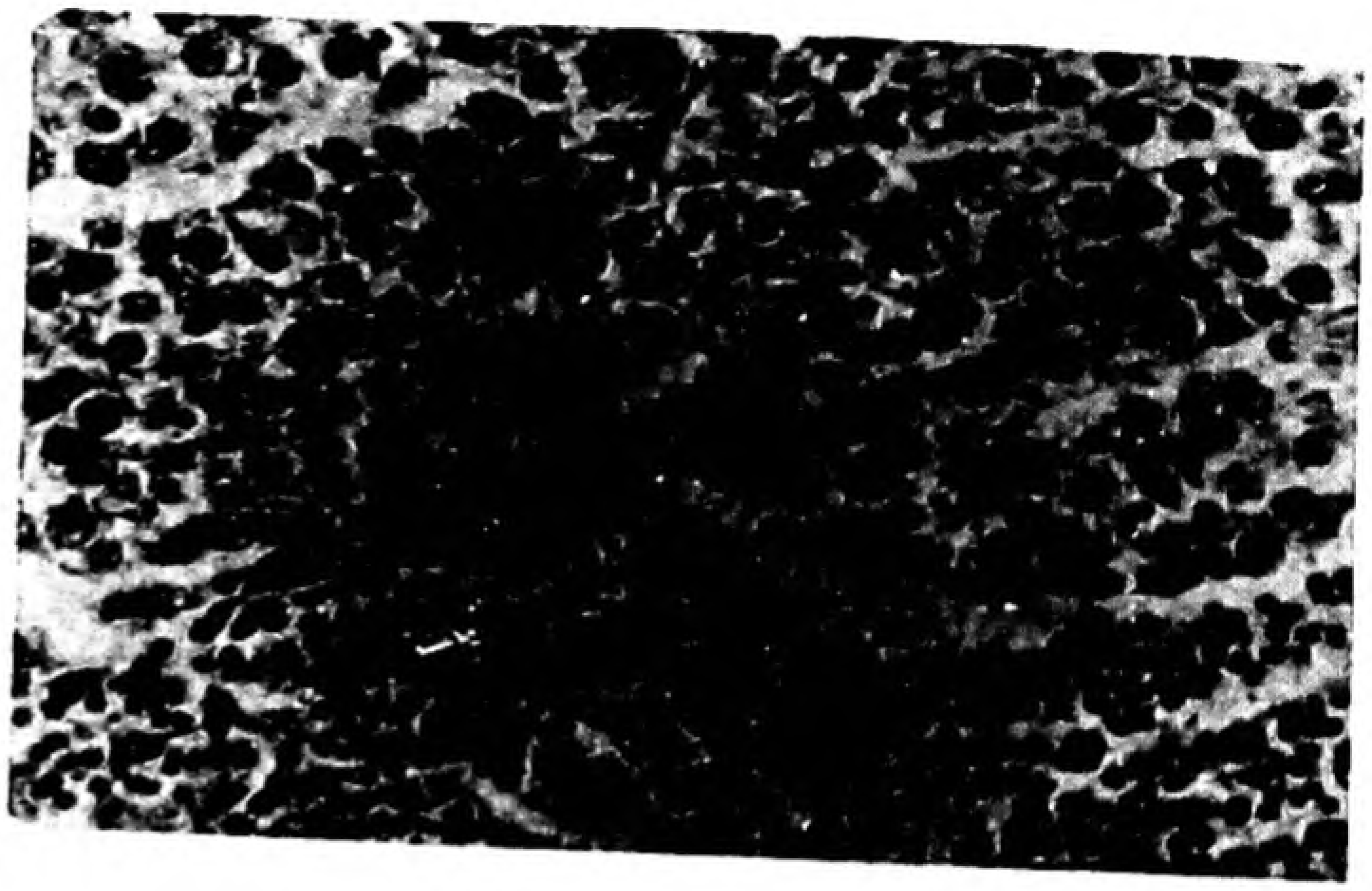

Fig. II Seminoma

Teratoma:

Arose from right undescended testis. tumor was 150x130x50 mm. Cut surface grey white with haemorrhagic areas intermixed with microcystic areas varying soft to firm in consistency.

Microscopy revealed heterogenous tumor components which included Keratinouscysts. respiratory epithelium. glandular spaces lined by tall columnar epithelium. immature cartilages. adipose tissue epidermis with sebacious glands. Immature neural elements were also seen locally.

Combined Germ Cell Tumor:

It developed in 20 years old male in right undescended testis. tumor 95x90 mm. confined by Tunica Albigunia. Testis was completely replaced by yellowish solid tumor alternating with microcystic spaces with mucinous feel. Areas of haemorrhage and foci of necrosis were seen.

Microscopy revealed combination of seminoma with Yolk Sac tumor in equal proportion.

Discussion:

13.4 percent testicular neoplasma in present study occured in undescended testis. Percentage of these tumors published in different series are those of Collins and Pugh[7] 5.9(58/952) Ahuja and Rao[8] 13.3 (6/45), Mackay and Seller[9] 4.4, Mostofi[5] 3.6, Gupta[10] et al 8, Cohen[11] et al 12.5 (7/51), and Reddy[12] DB et al 7(4/56).

Though our figures are higher than most of these studies but are closer to Ahuja et al and Cohen et al.

5 of 14 cases (28 percent) of present study occured bilaterally. This supports the observation of Gilbert and Hamilton who reported that patient with bilateral undescended testis. tumors in 24. 6 percent cases occurs bilaterally. 3 of 6 cases (50 percent) of Ahuja series occured bilaterally. Seminoma was the commonest tumor seen in present study which comprised 78.6 percent (11/14 cases). This supports the observation of Collins and Pugh who described seminoma to be commonest tumor in ectopic testis and their percentage being higher than scrotal testis.

In six of 14 cases tumor occured in right testis left side was involved in 3 cases and five cases had bilateral testicular neoplasma. This confirms the observation of Scully[13] that undescended testis occurs more commonly on the right side than on the left. This accounts for the preponderance of right sided seminomas.

Table - I: Clinical summary of tumor in undescended testis

Case No.	Biopsy No.	Age with	Clinical Presentation	Histological diagnosis	Additional remarks
1.	S-2974/84	30 BL	Lower Abdominal swelling	Seminoma	Wide spread metastases.
2.	S-1300/85	4 BL	Lump lower abdomin	Yolk Sac tumor	
3.	S-1429/85	? Rt	Rt. conguinal mass	Seminoma	
4.	S-1213/86	30 BL	Lump in lower Abdomin	Seminoma	Epitheloid granuloma
5.	S-510/87	35 Rt	Lump lower Abdomin	Seminoma	Infiltration of cord
6.	S-109/88	20 Rt	Rt. lower abodminal mass	Seminoma with Yolk Sac tumor	
7.	S-1776/91	25 BL	Lower abdominal mass	Seminoma	Epitheloid cell granuloma
8.	S-2076/91	25 Rt	Rt. Lower abdominal mass	Teratoma (mature and immature)	
9.	S-113/92	30 Rt	Rt. inguinal mass	Seminoma	Epitheloid granuloma
10.	S-205/95	40 L	Lower abdominal swelling	Seminoma	Para-aortic infiltration
11.	S-478/95	30 L	Left abdominal mass	Seminoma	Exuberant lymphocutic infiltrate with L.Follic
12.	S-15/9535 L	35 L	Lt. inguinal mass	Seminoma	
13..	S-1075/95	60 BL	Abdominal lump	Seminoma	
14.	S-1144/95	70 Rt	Abdominal lump with hemoptysis and cervical lymph hodes	Seminoma	Wide spread metastasis

B.L : Bilateral
Rt. : Right
L. : Left

Table - II

Undescended Testis and Occurance of Tumor

Series	UD/Total cases	Bilateral UD with bilateral tumor	Unilateral UD with unilateral tumor same side	Opposite side
1. Collins & pugh (1964)	58/952	11/11	47/37	9+1
2. Ahuja & Rao (1960)	6/45	3/3	3/3	-
3. Gupta et al	10/125	-	-	-
4. Reddy Bhaskara et al (1965)	4/56	-	4/4	-
5. Present study	14/112	5/5	9/9	-

UD (Undescended) + side unknown 1 case

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"A Study of Trace Elements, Copper and Zinc in Patients of Head, Neck and Oesophageal Malignancies."

Syed Javaid A. Bihaqi*; Mohd. Latif; Sheikh A. Ahad*****

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Abstract: A comparative study of trace elements copper and zinc along with impact of treatment in 50 patients of head, neck and Oesophageal malignancies is presented. Trace elements copper and zinc were estimated both in blood and tissues of the patients and control subjects using atomic absorption, photospectrometry. Substantial differences exist in serum and tissue concentrations of such elements in malignant and normal tissues. Statistically significant differences in pretherapy and post therapy, serum copper and zinc and copper: zinc ratio were also observed.

Key words: Malignancy, oesophagus trace elements.

Introduction

Biological markers are being utilized to assess host response to treatment in a variety of malignant diseases and serve as an important aid for effectively monitoring cancer therapy. Levels of myelo-immunoglobulin, human chronic gonadotropin, α fetoprotein and carcinoembryonic antigen are increasingly being estimated to assess the clinical course of the patients with a variety of cancers. Inspite of having modern technology and assistance, there is, still, a need to have some never but easy method of assessing the treatment response of cancer, so that different treatment modalities may be offered to the sufferer before the ill episode makes him uncurable.

The role of trace elements in malignant disease evokes increasing interest with the advent of accurate methods of detecting their presence in human tissues. The observation that substantial difference exists in the concentration of such elements as zinc, copper, magnesium and calcium in malignant and normal tissues, suggests that these trace elements may play a role in the etiology or metabolic consequences of cancer and thus excites interest in their possible use in diagnostic tests and assessing the response to different methods of treatment.

Material and Methods

The present study was conducted on 50 patients of head, neck and Oesophageal malignancies admitted in the Department of ENT at SMHS hospital Srinagar. A control group, comprising of 20 age and sex matched normal subjects, was also included in the study for the sake of comparison.

The diagnosis of the ailment was confirmed by histopathological examination of the malignant tissues which were obtained at the time of surgical procedures. In order to compare the results, control tissues from prescribed sites in head, neck and oesophagus were taken from patients, otherwise healthy, who underwent endoscopic and other surgical procedures for minor ailments like removal of foreign bodies.

Blood, from confirmed patients of malignancy before and after treatment, and subjects included in control group was collected. Trace elements, copper and zinc were estimated in both blood and tissues of the patients and control subjects, using atomic absorption photo-spectrometry.

Observations

The patients were divided in various groups, according to their site of involvement, which included 21 (42%) case of malignancy Oesophagus, 12 (24%) cases of malignancy larynx, 9 (18%) cases of malignancy pharynx, 3 (6%) cases each of malignancy para-nasal sinuses and neck lympnodes and 1 (2%) case each of malignancy oral cavity and submandibular salivary gland. 41 (82 per cent) patients were without secondaries and 9 (18 percent) cases had clinical evidence of secondaries.

The values obtained, in the present study, are shown in various tables.

Discussion

A number of investigations have provided evidence of errors in trace element metabolism in can-

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* *Registrar,* ***Assiant Professor*
****professor and Head*
Department of Otorhinolarngology (ENT)
Government Medical College-Srinagar-190 010
Kashmir (India)

TABLE I: Showing Serum tissue Cu & Zn in 50 patients of head, neck and Oxesophageal malignancy before treatment.

	Control (20)	Oesophagus (21)	Larynx (12)	Pharynx (9)	PNS (3)	Neck Nodes (3)	S.Gland (1)	Oral cavity (1)
Sr. Cu. Ug/100ml	124.21	178.11	173.96	191.39	182.56	206.96	128.03	143.04
Tissue Cu.ug/gr	62.32	132.43	130.69	133.89	94.13	140.13	152.00	129.00
Sr. zinc ug/100ml	98.23	81.50	77.23	75.92	86.13	81.07	59.05	91.02
Tissue zinc ug/gr.	262.8	1000.74	936.88	1344.03	1011.14	686.07	899.01	609.01
Sr.Cu:Zn	1.26	2.19	2.25	2.53	2.11	2.53	2.15	1.56
Tissue Cu:Zn	0.25	0.13	0.14	0.10	0.09	0.20	0.16	0.21

TABLE II: Table showing modalities of treatment offered to 45 patients of head, neck and Oesophageal Malignancies:-

Group:		Radiotherapy:	Surgery	Chemotherapy	Combined	Refused/Lost:
1. Oesophagus	(21)	18	X	X	X	3
2. Larynx	(12)	11	1	X	X	X
3. Pharynx	(9)	09	X	X	X	X
4. PNS	(03)	X	1	1	X	1
5. Neck nodes	(03)	02	X	1	X	X
6. Salivery gland	(01)	X	1	X	X	X
7. Oral Cavity	(01)	X	X	X	X	1
	50	40	3	2	X	5

TABLE NO III: Showing Sr. trace elements in 45 cases after treatment and impact of treatment.

Group:	No of cases:	Serum Cu ug/100ml	Sr. Zn ug/100ml	Sr.Cu:Zn ratio Postherapy	Sr.Cu.:Zn Pre-Therapy (Cases)
1. Control	20	124.21	98.23	1.26:1	1.26 :1 (20)
2. Oesophagus	18	125.97	92.31	1.35:1	2.19 :1 (21)
3. Larynx	12	132.03	90.18	1.46:1	2.25 :1 (12)
4. Pharynx	09	124.54	90.03	1.38:1	2.11 :1 (3)
5. PNS	02	147.02	87.35	1.68:1	2.11 :1 (3)
6. Neck nodes	03	111.56	87.63	1.27:1	2.53 :1 (30
7. Salivary gland	01	124.03	88.05	1.40:1	2.15 :1 (3)
8. Oral cavity	X	X	X	X	

cer. Many investigations have shown the difference in concentrations of metal containing enzymes between various types of cancers [1], while others have discussed the influence of metals like Zinc on RNA & DNA, and carcinogenic effect of metals and chelating agents [2,3]. The difference in the concentrations of metal containing enzymes between various types of cancers in mouse were shown by Green stain (1956). Weser and associates (1969) have discussed the influence of metals like Zinc.

Accordingly, the present study which was taken in hand in the department of otorhinolaryngology, SMHS Hospital, Srinagar, is an attempt to determine the behavior of trace elements, copper and Zinc, in patients of neoplastic disease of head, neck and Oesophagus.

The mean serum copper level, 124.21 ug/100ml obtained in 20 healthy individuals of control group, is in close approximation with the results obtained by various investigations [4,5,6]. The serum Zinc level 98.23 + 14.12 ug/100ml estimated in control group is also in conformity with the values reported earlier [6,7]. The mean tissue copper level 62.32 ug/gr and mean tissue Zinc value 252.8 ug/gr., obtained in control group, also correlates with the observations of other workers [6]

In the present study, significantly increased levels of SC were obtained in all the patients of head, neck and oesophageal cancer (P<.001) table 1.

No significant difference was found in the values of SCu in patients of head neck & Oesophageal malignancy with or without secondaries, the values being 178.58±48.95 in patients without secondaries, and 185.43±26.42 in case with clinical evidence of secondaries. The SCu obtained in the present study according to the site of tumor were 178.11 in oesophagous, 173.96 in larynx, 191.39 in pharynx, 143.4 in oral cavity, 182.56 in PNS malignancies, 206.,95 in Neck nodes & 128.3 in Salivary gland malignancy. Same observations have been reported earlier (6,8). Tissue copper level have been found increased in various body cancers(5,6,7). These observations of various workers are in agreement with the findings in the present study in which tissue copper level according to the site of tumor were 132.43 in Oesophagues, 130.69 in larynx, 133.89 in pharynx, 94.13 in PNS, 129.00 in oral cavity 140.13 in Neck node and 152.00 in salivary gland malignancy (table I) However there was no significant difference between the mean tissue copper values in patients with or without secondaries, the values being 131.15±19.09 in patients with clinical evidence of secondaries.

A statistically significant decrease in SZn as depicted in table I were observed in all the cases of head, neck and oesophageal malignancies included in the present study, as compared to SZn in normal control group (P<.001), in accordance to the observations of previous workers (6), being 81.50 in Oesophagus, 77.23 in larynx, 75.92 in pharynx, 91.02 in oral cavity 86.13 in PNS malignancy 81.07 in Neck nodes and 59.05 in salivary gland malignancy.

The tissue zinc levels obtained in the present study (Table I) were significantly increased in all the cases as compared to tissue zinc concentrations in normal control group (P<.001). In contrast to tissue copper level, a significant difference was observed between the tissue zinc values estimated in the patients without secondaries, (970.87±177.60) and tissue zinc content (1128.14±272.17) estimated in Patients with clinical evidence of secondaries. This finding was similar to that reported earlier (6).

As shown in table - I the pretherapy serum copper: Zinc ratio estimated in the present study, were markedly elevated as compared to copper; zinc ratio in control group (1.26). being 2.19 in Oesophagus, 2.25 in larynx, 2.53 in pharynx 2.11 in PNS 1.56 in oral cavity, 2.53 in Neck nodes & 2.15 in salivary gland cancer. The tissue Cu/Zn ratio in all the cases studied, were decreased as compared to normal tissue cu/zn ratio (0:25). The ratio obtained were 0.13 in Oesophagus, 0.14 in larynx, 0.10 in pharynx, 0.09 in PNS, 0.20 in neck nodes 0.16 in salivary gland malignancy .However, these ratios were in close approximation with each other in patients with or without secondaries, the ratio being 0.14 in patients without secondaries and 0.12 in patients with secondaries.

Different treatment modalities as shown in table II were offered to the patients included in the present study. The post-therapy serum copper and Zinc values were estimated in these patients after successful completion of the treatment.

The results are shown in table II. A statistically significant decrease in post therapy SCu was observed in all the cases as compared to the pretreatment SCu(P<.001). Same observation is being shared by many investigators (9,10,11,12).

However, no significant difference between preoperative and post-operative values of SCL has been reported in one of studies reported earlier (13)

In the present study, statistically significant increase in serum content of zinc after treatment was observed in all the case of head, neck and Oesophageal malignancies, as compared to pretherapy, serum zinc values. The findings of the present study are in accord with the earlier observations

(10,14). The post therapy serum cu,zn ratios, in this study, were decreased as compared to pretherpy ratios, in all the cases studied, coming almost towards the normal values, the ratios being 1.35 in oesophagus 1.46 in larynx 1.38 in pharynx, 1.68 in PNS, 1.27 in neck nodes and 1.40 in salivary gland.

Summary and Conclusion

In this study of 79 cases including 20 healthy controls and 50 patients of malignancy head, neck and oesophagus, a statistically significant elevation of SCu ($P<0.001$) and decrease in SZn ($P<0.001$) were observed in the patients of cancer as compared to control group. Both mean tissue copper levels and mean tissue zinc levels in all the cases of head, neck and oesophageal cancer were significantly increased as compared to mean tissue levels of copper and zinc in normal control group ($p<.0011$). The serum cu; zn ratio in all the cases of malignancies, studied, were markedly raised and tissue copper/zinc ratios were decreased before treatment, as compared to the ratios obtained in control group. However, the serum copper/zinc ratio in all the cases who received treatment, in different forms, decreased markedly reaching almost to values obtained in control group.

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Fine Needle Aspiration Cytology as an initial tool in the evaluation of Head and Neck Masses

M. Ashraf Darzi*#; Nisar A. Chowdri#;**
M. Akbar Bhat*#; G.M. Jan ****#**

JK Practitioner 1996; 3:4 245-248

***Abstract:** Four hundred and ten fine needle aspirations (FNAS) performed over a period of seven years on head and neck masses were studied for diagnostic accuracy and safety of the technique in comparison to surgical histological examination. Sixteen of the aspirates were unsatisfactory. Overall results in terms of accuracy, sensitivity, specificity, positive predictive value and negative predictive value were calculated as 96.44%, 93.2% 98%7% 98.05% and 95.41% respectively. FNAC missed 11 malignancies and 3 cases were wrongly diagnosed as malignant. No complication was associated with the technique. No radical treatment resulted from false positive results and no delay in treatment resulted from false negative reports. Being simple, safe and reliable, we recommend its wider use in the initial assessment of masses in the head and neck region.*

***Key words:** Masses, Head and neck; Biopsy, Needle.*

Introduction

Fine needle aspiration cytology (FNAC) has undergone gradual improvement since it was first introduced by Kun in 1847.[1] Martin and Ellis[2] introduced the technique for evaluation of head and neck abnormalities in 1930. In 1971 Engzell and colleagues[3] reported excellent results with FNAC. Since then FNAC has become increasingly popular and numerous reports have demonstrated its safety[3,4] and cost effectiveness.[3,4,5]

Diagnosis of swellings in head and neck region by purely clinical criteria is unreliable. Because of number of vital structures in this region, incisional/ excisional biopsy can be sometimes difficult and even hazardous procedure. Open biopsy of tumor mass can spread the disease to surrounding tissues with inoperable or fatal results[2]. Aspiration biopsy whereby core or fragment of tissue is removed for histopathological examination has a disadvantage of more local trauma and potential damage to adjacent structures.

Although many encouraging reports of FNAC for head and neck masses have appeared in the medical literature[6-14] it is still not widely practised and many patients with head and neck swellings are still subjected to hazardous and unnecessary open diagnostic surgical biopsies. The present study was undertaken with a view to demonstrate usefulness of FNAC on head and neck masses and commend its wider use as an important part of initial assessment for any swelling in this region.

Material and Methods

Four hundred and ten masses in the head and neck region were subjected to FNAC over a period of seven years. The technique used in this study was the same as described by Zajicek.[15]

The skin over the swelling is swabbed with an alcohol sponge. A 20 ml disposable syringe is held in a metal syringe holder (comeco) which enables the operator to perform aspiration with a single hand. The lump is steadied with the other hand and the needle introduced in different directions into the mass and the plunger withdrawn forcefully whilst moving the tip of the needle within the tumor. Before removing the needle, the plunger is released to equalize the pressure . A sample of the specimen is thus collected in the core of the needle and expelled onto the glass slides, smeared and air dried. The smear is fixed with 95% ethyl alcohol and stained with May Grunwald - Giemsa technique.

All the patients were later subjected to incisional/excisional biopsy for histological confirmation and the results compared with cytological diagnosis.

Results

A total of 410 FNACS were performed on head and neck masses. All aspirations were done on out-

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**Associate Professor and Head, **Consultant, ***Senior Resident, ***Professor,*
#Department of Plastic and Reconstructive Surgery, Department of Pathology.,
S.K Institute of Medical Sciences, Srinagar, Kashmir (India)

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patient basis without any anesthesia and without any imaging technique. Fifty four percent were males. The overall median age was 44.4 years (range 7 days to 78 years) and the median size of the mass was 6 cms.[2] Adequate material was obtained in 96.1% successfully.

Majority of the masses aspirated were lymph nodes (42.20%). Thyroid swellings were 31.22% and salivary glands 8.29%. Rest of the masses (18.29%) were classified as miscellaneous including jaw tumors, hemangiomas and other cutaneous and subcutaneous swellings.

The comparison of cytological and histological results is shown in Table 1. Opinion was not possible in 15 lymph node masses and one thyroid mass on FNAC.

The various parameters analysed statistically as per site of the lesion are summarized in Table 2.

There were no complications attributed to the procedure in this series.

Discussion

FNAC plays an important part in the initial outpatient assessment of all patients with head and neck massess. The patient history and clinical examination will usually provide differential diagnosis. However, in most cases cytological examination is necessary to arrive at a definitive diagnosis and determine further line of management.

Sixteen (3.9%) of our aspirates were unsatisfactory which is comparatively less as compared to Smallman et al[7] (15%), Schwarz et al[10] (9.3%) and Sismanis et al[11] (13.3%). The most common cause of unsatisfactory report is failure to obtain sufficient amount of cellular material. The number of unsatisfactory aspirates can be reduced by proper handling of the material and repeat aspirations. Opinion was possible in 18 lesions on repeat aspiration in our series.

The overall accuracy in our series was 96.44% which compares well with the results of other series reported by Frable and Frable[4] (95.9%), Meyers et al[16] (93.6%), Wilson et al[13] (91%) and Young et al[14] (94.5%). The overall results of sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPC) are also comparable with other published series (Table 3).

Among thyroid swellings, a major difficulty in cytological interpretation is distinguishing a hyperplastic nodule from follicular tumor and it is impossible to separate well differentiated follicular carcinoma from follicular adenoma on FNAC.[17] Indeed the distinction requires histological demonstration of capsular and vascular invasion by tumor cells.[18] Cytological criteria taken in conjunction with clinical findings hard consistency, presence of nodes, constitutional symptoms etc. can provide a high diagnostic yield of follicular carcinoma[19]. There were only 3 false negative results in 128 thyroid swellings in our series which is lower than that of Gardiner et al[18] (11.5%).

FNAC has proved to be of considerable use in the management of salivary gland lesions. Schwarz et al[10] has reported PPV of 100% and NPV of 94% in his series. O'Dwyer et al[20] reported sensitivity of 73% and a specificity of 94%. Frable and Frable[21] similarly reported sensitivity of 92% and specificity of 94% in salivary gland lesions. High accuracy was also observed in our series (Table 2). FNAC can provide more informed consent regarding the risk of damage to facial nerve, possible need for neck dissection and also determine urgency for scheduling surgery. The only false positive report of malignant change in pleomorphic adenoma in salivary gland swellings in our series was due to pleomorphism and atypia of the pleomorphic adenoma. The false negative results in parotid swellings are due to misinterpretation of pleomorphism of the epithelial pleomorphic adenoma.[22]

Of the 173 lymph node masses there were 2 false positive and 2 false negative results in our series. The diagnostic accuracy of metastatic carcinoma was 100%. Malignant lymphoma cannot be fully classified on aspiration cytology alone, although Caster et al[15] have reported a high degree of accuracy of FNAC in the diagnosis of lymphomas. Technique is useful in detecting recurrent disease and transformation of lympoma from small to large cell type.[10] FNAC is also of benefit in assessing benign lymphoid lesions but interpretation should be made in the light of clinical situation. The false positive diagnosis is due to difficulty in interpreting atypical cellular architecture of the chronically inflamed nodes.[13] False negative rate of 13% was reported by Schwarz et al.[10] No opinion could be given for 15 lymphnode masses in our series but false negative diagnosis was made only in 2 patients. This is either because of aspiration of necrotic material or misinterpretation of cells as atypical chronic non-specific inflammatory nature. The cytomorphologic features typical of reactive lymhadenitis and the diagnostic pitfalls have been recently discussed Stani.[23] If a malignancy is suspected in lymph node lesions a negative cytology report must not be considered sufficient.

FNAC was found to yield high accuracy rate of 96% in miscellaneous group of swellings with only 3 false negative results including squamous cell

Table 1 Showing distribution of lesions and comparison of cytological and histological diagnosis

	Locations		FNAC	Histopathology	
				B*	M**
1.	Thyroid	B	87	84	3
		M	40	0	40
2.	Salivary Glands	B	18	15	3
		M	16	1	15
3.	Lymphnodes	B	86	84	2
		M	72	2	70
4.	Miscellaneous Group	B	49	46	3
		M	26	0	26

B* Bengin

Table 2 Statistical parameters calculated after comparing FNAC and histological results for various head. & neck massess

	Statistical Value %	Thyroid %	Salivary gland %	Lymph nodes %	Miscell-aneous %	Over all %
1.	Accuracy	97.63	80.23	97.46	96.00	96.44
2.	Sensitivity	93.02	83.33	97.22	89.66	93.20
3.	Specificity	100.00	93.75	97.67	100.00	98.70
4.	Positive predictive value	100.00	73.75	97.22	100.00	98.05
5.	Negative predictive value	96.55	83.33	97.67	93.87	95.41.

Table 3 Over all results in comparison to other studies

	Author	No. %	Sensitivity	Specificity %	PPV %	NPV %
1.	Frable & Frable (1979)[4]	567	95.20	98.70	98.40	96.20
2.	Sismanis et al (1980)[11]	107	90.30	100.00	100.00	84.20
3.	Young et al (1981)[14]	513	91.50	97.10	- -	
4.	Scheider et al (1984)[9]	80	93.50	90.90	93.50	90.90
5.	Smallman et al (1988)[7]	120	98.81	94.44	93.65	97.14
6.	Schwarz et al (1990)[10]	182	92.00	100.00	100.00	89.00
7.	Present series	410	93.20	98.70	98.05	95.41

carcinoma, angiofibroma and one case of anaplastic carcinoma. There was no false positive diagnosis in this group.

No radical treatment resulted from false positive diagnosis and no delay in treatment resulted because of false negative reports. There were no complications in our series. The complication rate of zero or minimal has been reported by many authors.[4,8,11] Although there have been rare reports of tumor seeding with transperitoneal aspiration and it may be possible though rare with true cut biopsy, this has never been reported with FNAC.[3,22]

Cost effectiveness of the FNAC has already been reported in the literature[6,21].

From this study, we conclude that FNAC is simple, safe, quick, cost effective, repeatable and reliable diagnostic procedure that can be done on outpatient basis with minimal discomfort to the patient. There fore we recommend its routine use in the initial evaluation of a patients with head and neck masses.

Correspondence:
Dr. Mohammed Ashraf Darzi
Associate Professor & Head,
Department of plastic & Reconstructive
Surgery S.K. Institute of Medical Sciences
Soura, P.O. Box 27

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Experimental Medicine:

"Lithium and Kidney"

Shah, M.S.; Hassan, G.***

JK Practitioner 1996; 3:4 249-250

Abstract: *The present study evaluated the microscopic changes in kidney of albino rats, fed lithium. A total of 30 animals were divided into A and B groups. Ten animals in former group served as control, whereas twenty animals in group B were fed with lithium carbonate for a period of 90 days. The microscopic study of kidneys in drug treated animals revealed degenerative changes in proximal and distal convoluted tubules. Besides a marked interstitial fibrosis with atrophy was also evident.*

Key words: *Lithium, kidney, tubular, degeneration.*

Introduction

Lithium was introduced in medical practice after its calming effects were noted by Cade; F.J in 1949.. Later renal histopathology after lithium therapy was extensively investigated since it is mostly excreted in urine..Experimental studies conducted by Radomski et al in 1950 and Schou; M in 1958 on dog and rat indicated that kidneys are primarily effected. They observed changes of degenerative nature in tubular epithelium of kidneys. In 1979 Hensen et al reported that patients suffering from interstitial nephropathy after long term treatment with lithium showed an increased fibrotic tissue in the medulla and cortex.

Material and Methods

Thirty albino rats weighing 150 gms on an average were used during present study. The animals were categorised in A and B groups. Ten animals in former group received usual food and plain water and served as a control, whereas twenty animals in group B in addition to usual food and drinking water received lithium carbonate in a dose of 4 mgs/ 100 gms of body weight mixed with flour in the form of pellets. The process of drug administration continued for 90 days regularly. The tissues obtained were processed manually for block making. Blocks were subjected to routine technique and slides thus prepared were stained with haematoxyline and eosin for microscopic study.

Observations

Microscopically, the kidneys treated with lithium showed degenerative changes in proximal and distal convoluted tublues.

Besides a marked interstitial fibrosis with atrophy was also evident (Fig. I).

Discussion

Post lithium renal histopathology has been extensively investigated since it is mostly excreted in urine. Radomski et al (1950) observed degenerative changes in tubular epithelium of kidneys. Similar changes were also reported by Andrew; PE and Dwayne; A, Ollerich (1972). In their study Hestbech; J et al (1977) observed tubular atrophy, interstitial fibrosis and sclerotic glomeruli, identical with observations of Lavender et al (1973) and Londop; et al (1975). In 1978 Burrows; et al reported tubular atrophy and interstitial fibrosis, coincidental with observations of Hestbech, J et al (1977). Renal tubular damage and interstitial oedema were also described by Chapman and Lewis (1972) which was in accordance with the earlier reports on dog by Radomaski et al (1950) and on rats by Evan and Ollerich in 1972.

In present study also a tubular degeneration and marked interstitial fibrosis with atrophy were observed, thus identical with the findings of Radomski et al (1950). Andrew, PE and Deayne; A. Ollerich (1972). Hestbech; J et al (1977) and those of ||Burrows, et al (1978). It is obvious that prolonged lithium administration induces renal damage and in view of histological observations made during present study it is suggested that renal functions should be monitored in those who are on long term lithium therapy.

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*Professor and Head, **Demonstrator
Department of Anatomy.
Govt, Medical College. Srinagar, Kashmir. (India)

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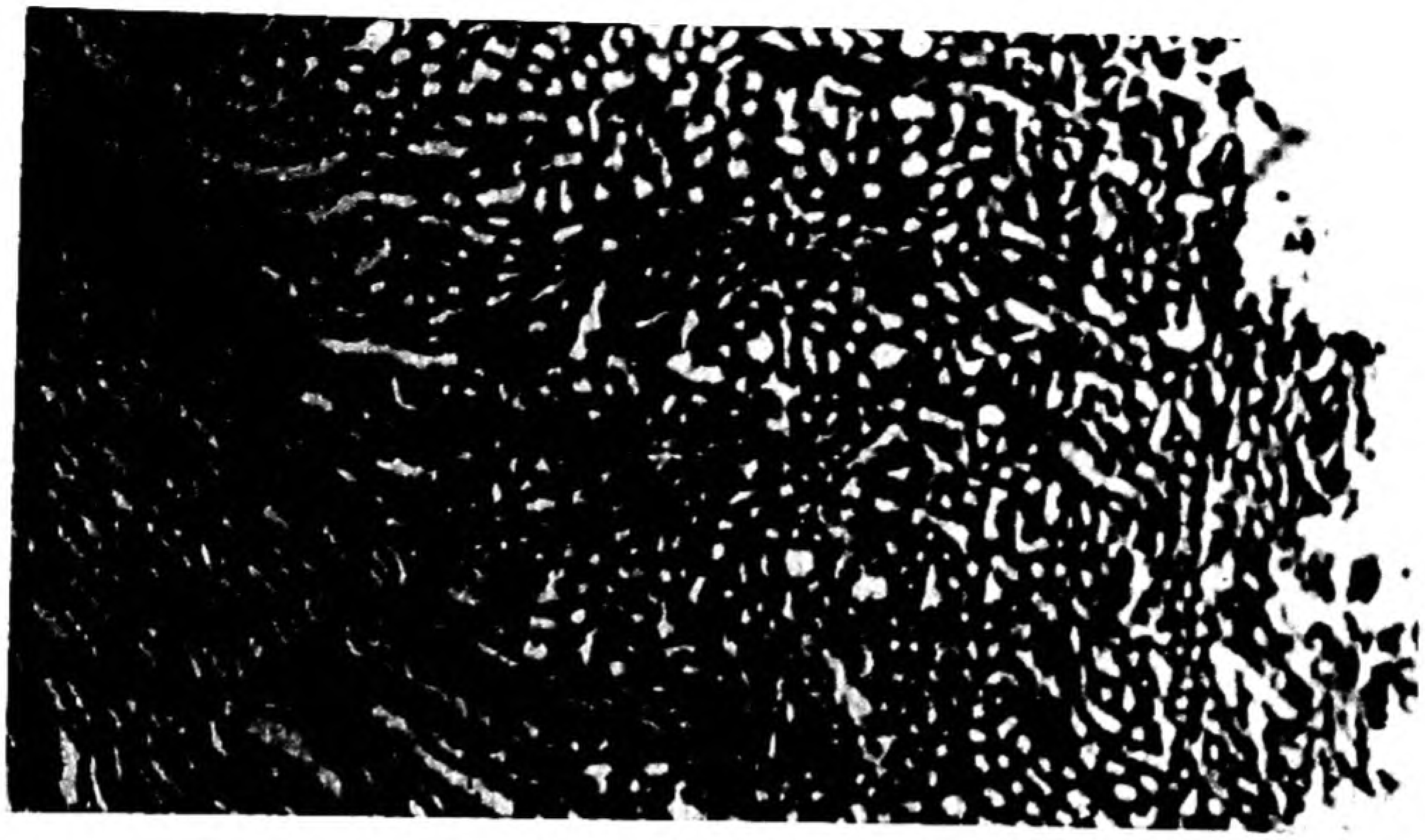

Fig.I: Microphotograph showing kidney with markea intestitial fibrosis (I.F) with atrophic (Atr.) tubules in group B animal during 12th week. Stain H and E Magnification: 10x10 x

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Case Reports:

"Clouston Syndrome"

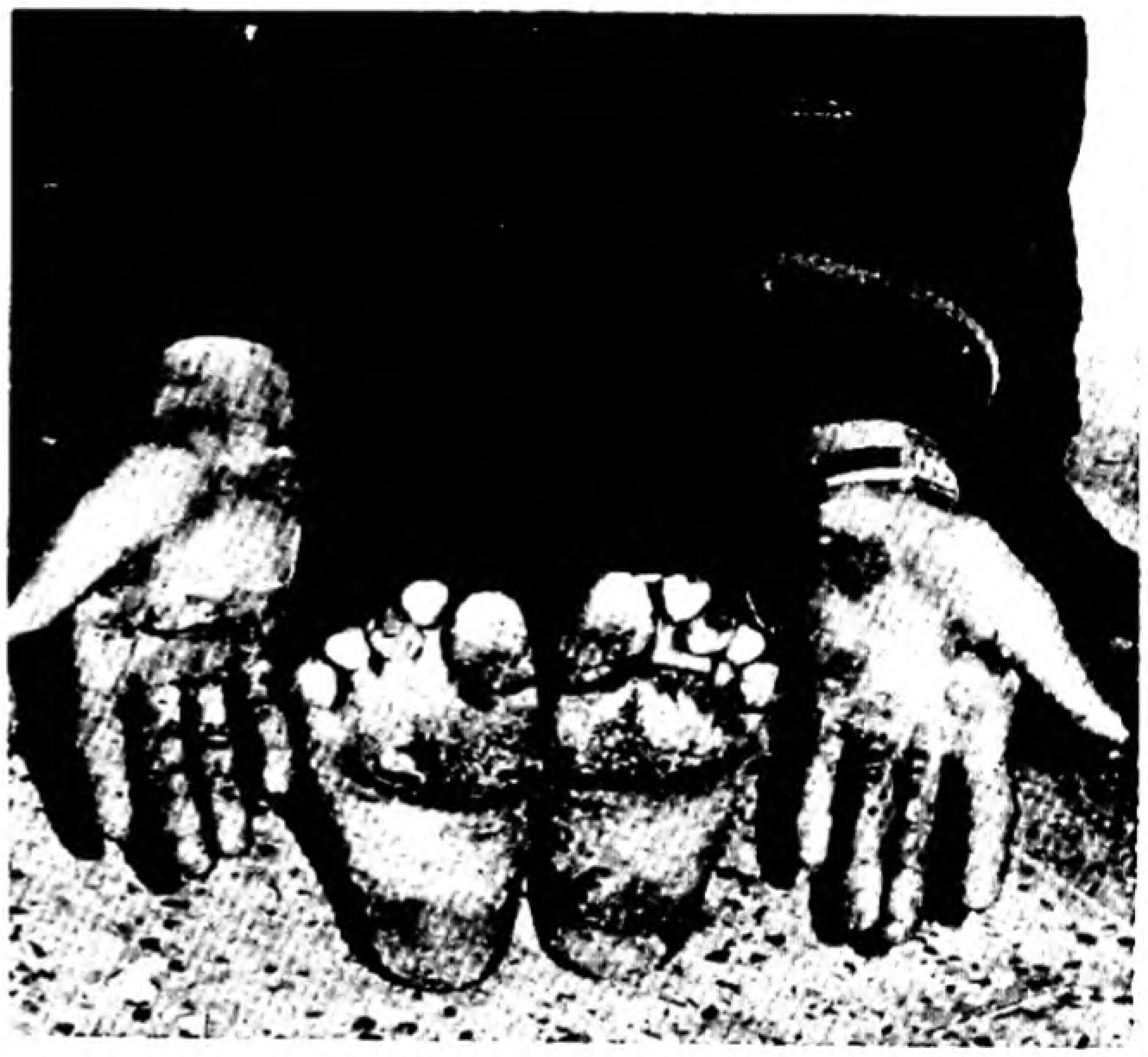
Qazi Masood*; Sheikh Manzoor;**
Aftab Ahmad***

JK Practitioner 1996; 3:4 251

***Abstract:** Clouston syndrome (Hidrotic ectodermal dysplasia) is a rare syndrome characterized by nail dystrophy associated with defects of the hair, and keratoderma of palms and soles and oral white lesions. Here we report a case of this rare entity.*

Case Report

A 20 year old boy presented to us with generalised palmoplanter thickening, and nail dystrophy. Both these changes were noticed from early childhood by the patient. His physical examination revealed, palmoplanter keratoderma, with dystrophic nails (Fig I). Besides this, hair were sparse and brittle and fine. His oral cavity showed multiple whitish lesions on hard palate. Rest of his body was normal.



Discussion

Clouston syndrome is a rare autosomal dominant disorder characterised by palmoplanter hyperkeratosis, hair defects, nail dysplasia and oral white lesions.[1]

The key feature of this syndrome is the nail dystrophy. In about 30% patients there may be no other abnormality[2]. In it's complete form the scalp hair is very sparse, fine and brittle or completely lacking. The teeth may be normal. There are oral white diffuse lesions on palate[2] tongue. Plamoplanter hyperkeratosis may be seen.

It has been suggested that there is a molecular defect of keratin[3] Hyperkeratosis resulting from reduced degeneration is most evident on palms and soles. Biophysical and biochemical studies of the hair give findings consistent with the hypothesis that there is a depletion of matrix protein and disruption of or a failure to form disulphide bonds in the remaining keratin. Ultrastructural studies of hair have shown disorganization of hair fibrils with loss of the cuticular cortex.[4,5]

Our case report is a complete form of the clouston syndrome showing nearly all features including nail dystrophy, palmoplanter, keratoderma, hair changes and oral white lesions.

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** Assitant Professor . **Senior Resident*
****Registrar*
Department of Dermatology
Govt Medical College, Srinagar, Kashmir 190 010.
Correspondence to Dr. Qazi Masood

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Late Metastasis of Malignant Melanoma to Bladder A Case Report and Review of Literature

A.A. Bhat*

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Case Report

A 70 year old male referred by his General Practitioner with a few months history of post-micturition dribbling and microhaematuria. History included wide excision of a malignant melanoma followed by skin grafting from his left arm 8 years before. for which he had been reviewed regularly. He also suffered from chronic obstructive airways disease and glaucoma.

Examination confirmed moderate enlargement of benign feeling prostate. with an obstructive flow pattern on flow rate. All the routine investigations for haematuria, including I.V.U., were normal. Cystoscopy revealed many black solid looking tumors about half a centimetre in diameter on the posterior wall of the bladder. Resection biopsy of the lesions was performed. and histology confirmed the presence of malignant melanoma.

Abdominal CT scan was normal, but CT of the chest showed metastases throughout both upper lobes. even though a plain X-ray chest was clear. The patient refused palliative chemotherapy. Two months later brain metastases were confirmed by CT scan when the patient was admitted in an acute confusional state. The condition. however, improved on Dexamethasone.

Comment

Melanomas comprise about 1.3 - 2% of all malignant tumors. Genito-urinary malignant melanomas account for less than 1% of all melanomas. Metastatic melanomas of the bladder are more common than primary ones. and less than 100 cases of metastatic melanoma of the bladder have been reported in the literature. Genem and Batal in 1956 presented a collection of 80 cases of secondary tumors of the bladder. and found 18 cases of metastatic melanoma which was second in frequency to only gastric carcinoma (25 cases).

The earliest case of metastatic melanoma of the bladder was reported in 1891 by Target. Several autopsy series in patients with malignant melanoma including that of Sheehan & Associates in 1963 (5,200 autopsies) have shown that the incidence of bladder metastases is 14 - 22%

The diagnosis is usually established by cystoscopic biopsy or at autopsy, and a thorough evaluation of the patient is done to look for other visceral metastases because the bladder metastases is usually a part of the widespread disease. Thus radical surgery is not indicated, and the tumour is both chemo. resistant as well as radio resistant. Prognosis is uniformly poor. The interval after the primary melanoma is usually less than 3 years for metastases to the bladder with the longest intervals encountered being 9 years. An interval more than six months free of disease statistically has a better prognosis. Most patients die of generalised disease within a few months after the diagnosis of metastases to the bladder is made, and the longest reported survival to date has been 3 years from the first definitive bladder biopsy. Such survival, however, is exceptional.

Correspondence to:
Dr. A.A. Bhat, MB; FRCS
Block B, Flat 11, Queen's Hospital,
Burton upon Trent, De13 ORB
Tel 01283 566333 (bleep 231)

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*Registrar. Department of Urology
Queen's Hospital. Belvedere Road.
Burton Upon Trent (U.K)*

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Recurrent Ameloblastic Fibroma with Atypical Presentation

Nisar Ahmad*; Mohd Ashraf Darazi;**
Shabir Iqbal*; Zuhair Afzal Khan******

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Abstract: The article presents a fibroma following hemimandibulectomy and radiotherapy. The patient is recurrence free 18 months following adequate excision.

Key Words, Ameloblastic fibroma, Recurrence, Treatment.

Introduction

Ameloblastic fibroma is a rare jaw tumor. Regezi and associates[1] reported that ameloblastic fibroma comprised 2 percent of 706 odontogenic tumors. Approximately 60 cases have been reported in the medical literature[2]. Recurrence rate is high if inadequately excised[3]. We report a case of large recurrent ameloblastic fibroma with atypical presentation.

Case Report

A 16 year male patient presented with an ulcerated painless swelling in the preauricular region with repeated episodes of bleeding. Two years back patient had undergone right hemimandibulectomy for slow growing swelling in the jaw with provisional diagnosis of ameloblastoma. 8 months later, an ulcerated swelling was noted in the preauricular region which was subjected to external radiation. Tumor responded and regressed completely. A year later swelling re-appeared. This time tumor was found to be more aggressive and increased in size over a period of 4 months when patient was first seen by us. Except for anemia, general physical and systemic examinations were normal. Local examination revealed a large fungating mass in right preauricular region and temporal region extending subcutaneously upto lateral third of forehead anteriorly and interiorly to angle of mouth (Fig. I). It would easily bleed on touch. Skin over subcutaneous portion of the tumor was free. The growth measured about 25cmx15cms. The growth had restricted mobility on the underlying tissues. Edge biopsy revealed ameloblastic fibroma. Tumor was excised widely in toto and the defect closed by trapezius myocutaneous flap, part of which got necrosed which was lateron covered by split thickness skin graft. Patient is under our follow up for last 18 months and is recurrence free (Fig. II).

Discussion

Untill recently, the ameloblastic fibroma was considered to represent a fairly non-aggressive, benign odontogenic tumor[4,5] simple enucleation and curretage was reported to be adequate treatment, [3,5] but Trodahl [3] has reported a high recurrence rate of 43.5 percent. Some instances of rapid recurrence (behaving more like an ameloblastic sarcoma) after adequate excision have been reported and enblock resection has been recommended for such lesions. [6] Radiotherapy for recurrent growth after hemimandibulectomy did help our patient in arresting the growth of the tumor temporarily but it was followed by a more aggressive growth measuring about 25cm x 15cm in contrast to tumors ranging from 1-8cms in diameter in one of the largest series reported by Trodahl.[3]

Although Elis and Fonseca[6] do not recommend resections routinely due to rarity of the more aggressive ameloblastic fibroma, many authorities are of the opinion that the lesion may be some what more aggressive than was originally thought and recommend excision with adequate margins and not simply surgical curretage.[7] We also feel that adequate excision rather than enucleation and curretage or radiotherapy should be the treatment of choice for these tumors.

The tumor may be mistaken for ameloblastoma on histological study, but the loose primitive appearance of the stroma differs markedly from the mature fibrous connective tissue stroma seen in the ameloblastoma.[8] In fact our patients was wrongly labeled as ameloblastoma initially, so accurate diagnosis is mandatory and to avoid missing a malignant change in the tumor, thorough histological examination of such lesions should be carried out before treatment.

Correspondence:
Dr. Nisar Ahmad Chowdri, MS; FAIS
Consultant, Department of Surgery
Institute of Medical Sciences, Srinagar
Kashmir (India) Pin - 190 011, Post Bag No. 27
Tele 0196-52509, 75617-19 Ext. 260

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* *Consultant,* ** *Professor and Head*
****Assistant Professor,* *****Sr. Resident*
Department of plastic & Reconstructive Surgery Institute of Medical Sciences, Srinagar Kashmir (India)

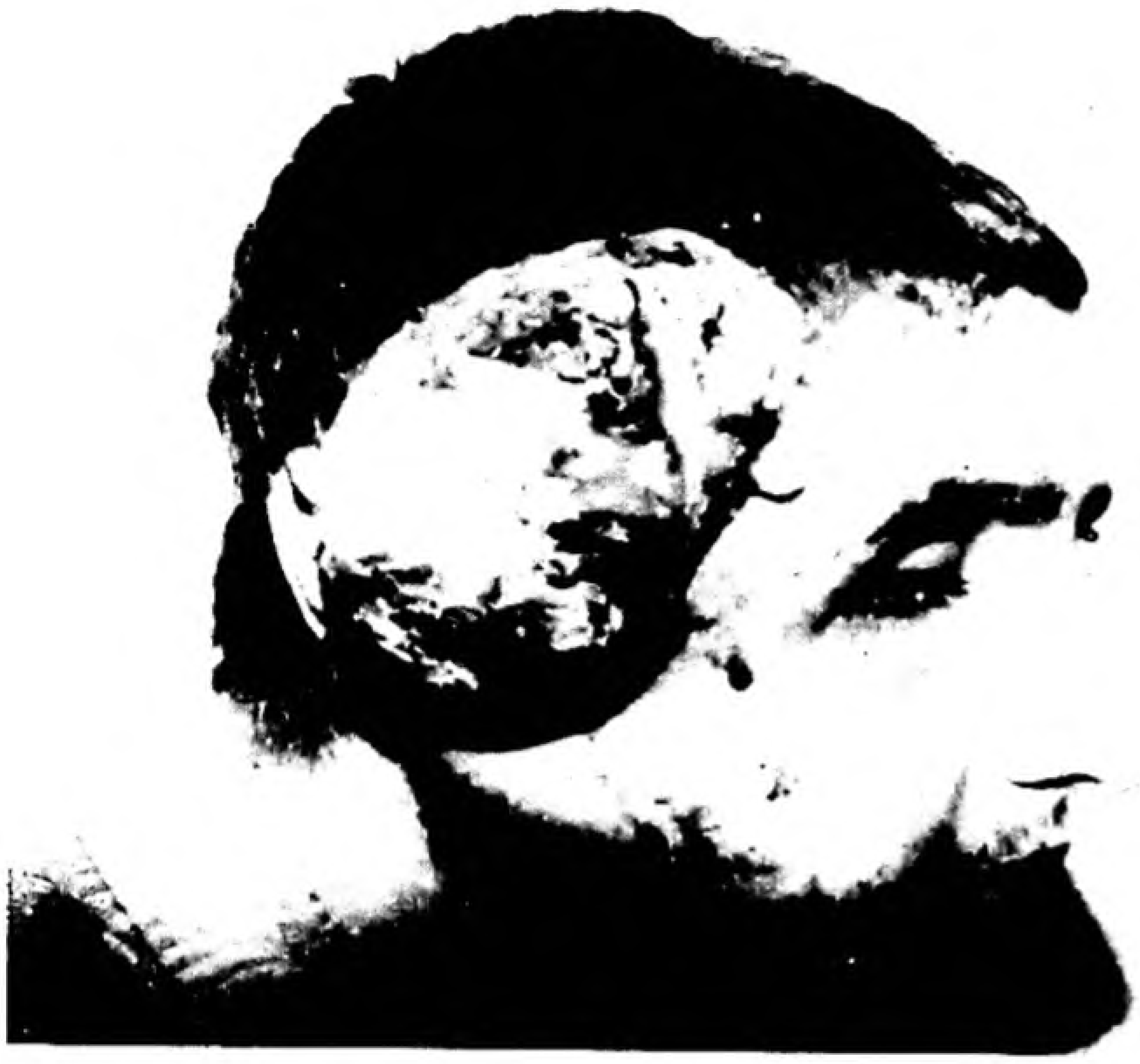

Fig. I: Recurrent ameloblastic fibroma after hemimandibulectomy and radiotehrapy.

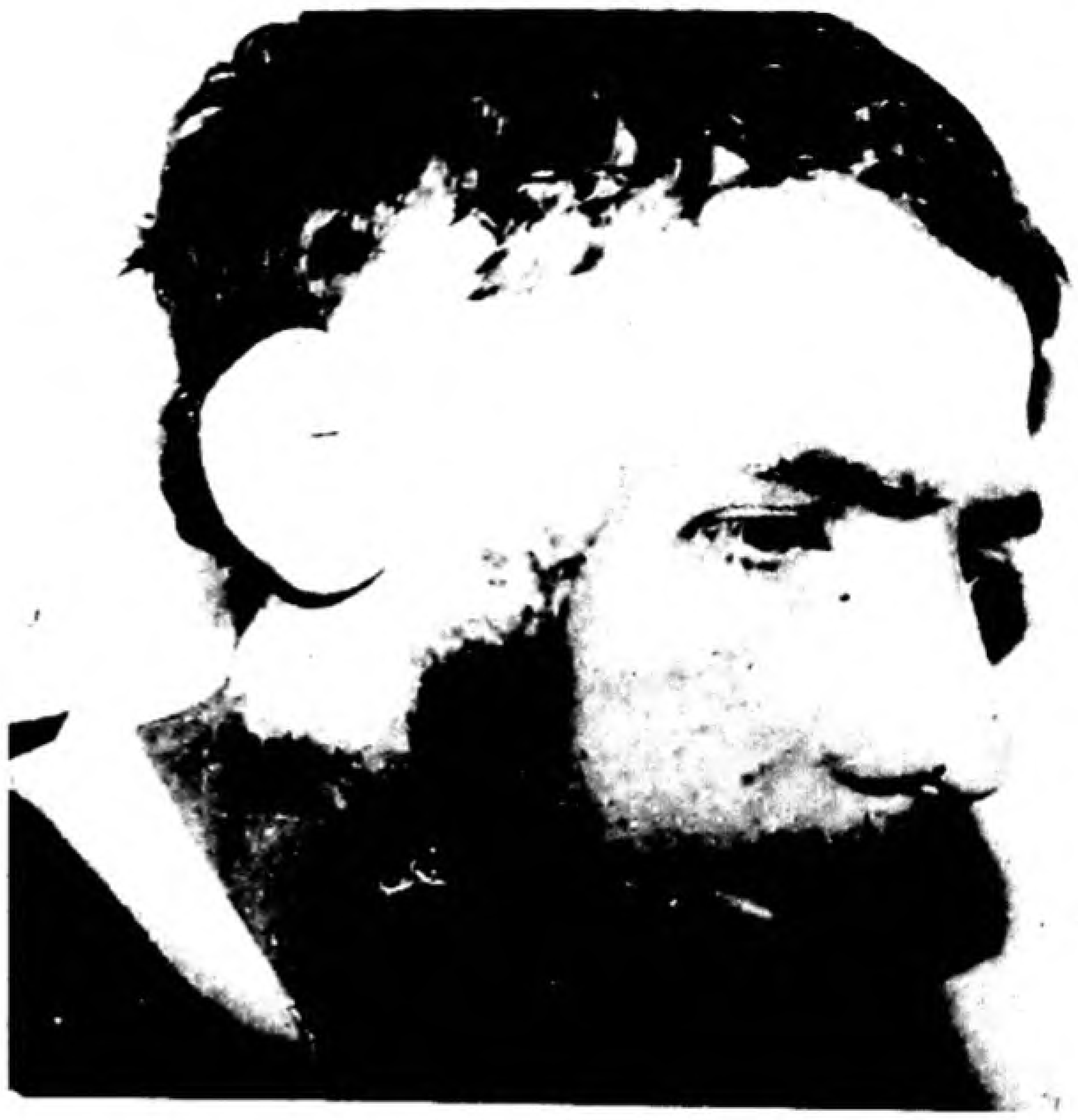

Fig. II: 18 Months after wide excision and reconstruction by trapezius myocutaneous flap.

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Special Article:

Islamic Teachings and Cancer Prevention "Alcohol and Cancer"

Mohammed Ali Albar*

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Introduction

Alcohol dependence is a world wide problem, more intense and widespread than other drug dependencies combined, with the exception of tobacco. In all countries were alcohol drinking is legally permitted, the majority of the adult population drink with some 10 present suffering from problems related to alcohol. (1-4) The 32nd World Health Assembly 1979, resolution 32,40 declared that "Problems related to alcohol and particularly its excessive consumption, rank among the world's major public health problems and constitutes serious hazards for human health, welfare and life.(4) The annual death toll due to tobacco, alcohol and other illicit drugs for U.S.A. is as follows:(5-9)

	U.S.A.	U.K
Tobacco	350.000	100,000
Alcohol	125,000	40,000
Drugs	6,000	159
Population	245,000,000	55,000.000

Tobacco smoking is responsible for 30-40 percent of total cancer mortality while alcohol drinking is responsible for 3-4 percent. (10,11) The confounding and synergistic action of both drugs make it difficult to pinpoint the specific role of each, since most heavy drinkers are also heavy smokers. (12-17)

Alcohol drinking is directly related to cirrhosis of the liver, the more the consumption of alcohol per capita in a country, the greater the number of liver cirrhosis cases and hence its mortality, and malignant change. (1-8)

The Role of Alcohol in Carcinogenesis

In studies of alcoholics, cancer is one of the major causes of increased mortality and morbidity (10,18) and in epidemiological studies alcohol is the second leading specifically identified cause of cancer, exceeded only by tobacco.

Alcohol acts a co-carcinogen and promoter; and alcoholic beverages contain many carcinogens e.g., polycyclic hydrocarbons and nitrosamines.(19) The Ames mutagenicity test is positive for a variety of liquors denoting the presence of many carcinogens.(20,21) Alcohol per se is not an initiator of tumours, but it acts as a cocarcinogen. Kustasune and co workers showed that benzopyrene, a known carcinogen, did penetrate mouse esophageal mucosa better when dissolved in alcohol than when dissolved in olive oil.(13) Another mechanism is through increased microsomal enzymes activity induced by alcohol ingestion which in turn enhance carcinogen activation.(22) Alcohol was also identified as a turnover promoter, both in animal models and epidemiologic studies.(23-25) The prolonged association of cancers with alcoholic cirrhosis of the liver is consistent with the action of alcohol as a tumor promoter, whereby these cancers tend to occur at sites of direct contact with ingested alcohol (mouth, pharynx, esophagus) or known sites of alcohol detoxification (liver).(22,26) Many studies have shown that heavy alcohol consumption is associated with heavy cigarette smoking. The alcoholic also suffers from malnutrition and poor dentition, all these factors play a role in carcinogenesis.

Despite these problems and many others, many studies demonstrated the relationship between alcohol and cancer. Prospective studies of heavy drinkers and alcoholics show an increase of about 70 percent in death from cancer.(18,27,28) The majority of these deaths are related to the strong association of heavy drinking with heavy smoking. In a series that used contemporaneous controls matched for smoking history, Klatsky found a 40 percent excess cancer mortality in a group of heavy drinkers.(10)

Epidemiologic studies shown an increased risk of cancer with increasing exposure to alcohol. There is a logarithmic rise in cancer esophagus with increasing alcohol exposure.(29) Similar studies of upper aero-digestive tract cancers confirm the dose response relationship.(13-17)

Alcoholic beverages contain many carcinogenic substances. The type of beverage however is of little importance compared with the amount of alco-

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***FRCP, DM (London)**
Consultnat of Islamic Medicine
King Fahd Medical Research Centre
King Abdulaziz University, Jedha (KSA)

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hol(13,16,30) with the exception of apple brandy which has been implicated in the high incidence of esophageal cancer in the Normandy region of France and red wine implicated in cancer of the stomach.(21,29,31,32) However, there is strong synergism between alcohol and tobacco, the first being a co-carcinogen which dissolves tobacco carcinogens and hence increasing mucosal concentration of these substances.(13) For laryngeal cancer there is 50 percent more risk of cancer with combined exposure than the summation risk of both substances.(33)

For esopharyngeal cancer, Rothamn estimates that 43 percent of the disease can be attributed to alcohol, 33 percent to smoking, and the rest being to the synergistic action of both substances.(34,35)

SPECIFIC SITES

Head and Neck

The association of squamous cell carcinoma of the head and neck with excessive drinking of alcoholic beverages was first noted in the 19th century.(13,34) Wynder demonstrated a ten fold increase of laryngeal cancer in people who drank more than 7 ounces of whisky daily. Subsequent case-control studies have shown similar results in oral, pharyngeal and laryngeal carcinomas. The effect of alcohol in these cancers is not due to a confounding effect of tobacco, alcohol is as important as tobacco, other etiologies factors e.g., poor dentition, hot beverages, deficiency of vitamins A and C, asbestos are less important than alcohol and tobacco.(36-41) Case control studies of the risk factors for carcinomas of tongue, gum floor, and other specified parts of the mouth were strongly linked with tobacco smoking and alcohol (wine, malt & other hard liquors).(41-43)

Esophagus

Esophageal cancer has been linked to tobacco smoking, chewing and alcohol intake. In American and European studies, alcohol has long been recognized as an important etiologic factor.(17,44-47)The incidence of cancer esophagus in Europe is greatest in France, the varying rates in that country are proportional to alcohol intake.The relative risk is 18 in those who drink more than 10 units daily, and if they smoke 20 cigarettes a day the risk is 44 fold non smoker teetotaler.(6)

The relative risk for esophageal cancer rises logarithmically with increasing use of alcohol and it is dose related. For heavy drinkers the risk factor is 20, even after correction for tobacco use.(29)For Western countries, about 80 percent of esophageal cancer are attributed to alcohol and tobacco abuse. (6,7,13,29,45)

There are other known causes of esophageal caner e.g., iron deficiency (Plummer-Vinson syndrome), ingestion of mate, corn or wheat based diets, deficiencies of riboflavin, nicotinic acid and zinc, and consumption of very hot drinks and foods.(13-29,45-47)

The high incidence of cancer esophagus in China is not related to alcohol consumption.(47) Similarly the high incidence in Saudi Arabia is not related to alcohol, whereby alcohol is proscribed and a very small percentage of the population drink secretly or when going abroad. Other factors e.g., iron, dietary deficiencies and consumption of very hot drinks and foods are blamed.(48)

Stomach

There is association between tobacco smoking, alcohol drinking and gastric carcinoma.(13,32,49) Flamant and McDonald found that cancer of the Cardia is more related to alcohol than other parts of the stomach.(50) Wine consumption in France is particularly related to gastric carcinoma, a high risk of 6.9 with heavy wine drinkers compared to non drinkers.(32,50,51)

Pancreas

Alcohol is an important cause of acute and chronic pancreatitis in Western countries. in some series there is a high incidence of carcinoma of the pancreas.(6,52,53)

Rectum Cancer

Population statistics shown an association between consumption of beer and rectal cancer in the U.S.(54,55) McMichael and Potter found similar association in the United States, United Kingdom, Australia and New Zealand, whereby beer consumption was related to colorectal cancer.(56,57) Data from the Third National Cancer Survey also supports the association between alcohol and colorectal cancer.(49) A study of irish brewery workers showed an increased risk of rectal cancer of 180 percent.(58) A similar study in Denmark showed a modest increased risk.(59)

Nevertheless the role of fat and low fiber seems more important in causation of colrectal cancer than alcohol.(17)

Liver

Liver damage is common sequelae of alcohol ingestion. The alcohol consumption per capita in a country is directly related to liver cirrhosis. In France and Italy where the highest per capita alcohol consumption is found in Europe, the incidence of liver cirrhosis mortality is highest (45-50 per 100,000 population annually).(4-8,60) Some 10-15 percent of those suffering from liver cirrhosis will develop liver cancer. (6,8) However, autopsy findings show a much higher incidence (30 percent) of hepato cellular carcinoma in alcoholic cirrhosis patients.(61,62) In a retrospective veterans administration study, Keller found a 30 fold increase in the relative frequency of liver cancer in those suffering from alcoholic cirrhosis compared with those without cirrhosis.(63)

Hepatocellular carcinoma is strongly related to hepatitis B virus, the malignant change being facilitated by the hepatotoxic effects of alcohol.

Cancer of the Breast

There is some increased risk of breast cancer associated with alcohol and cigarette smoking.(67) the relation with high intake of fat of animal origin is even more strongly related with breast cancer.(68)

Cancer of the Lung

This is increased in heavy drinkers of alcohol by 50 percent.(10) However many experts deny this association.(13,45)

In Conclusion

Heavy smoking and heavy drinking are linked with many cancers. There is strong synergism between the carcinogenic and cocarcinogenic substances in these two addictive drugs. Aerodigestive cancers features prominently in this respect. Alcohol is also associated with cirrhosis of the liver and liver cancer. It is also associated with cancer of the stomach, Pancreas and tenuously associated with breast cancer and lung cancer. Primary prevention will call for abstention from intake of both tobacco and alcohol.

Islamic teachings clearly prohibit alcohol intake. The Holy Quran has emphatically prohibited alcohol intake since 1400 years. "O ye who believe. Intoxicants and gambling, dedication of stones and divination by arrows are an abomination. The Satatan wants only to cast among you enmity and hatred by strong drink and games of chance, and to turn you away from prayer. Will you not then desist?"(69) The response was very remarkable indeed. The whole community of Medina cried " O Allah, we have desisted." Since then Muslim communities are the least affected by alcoholic beverages (AIKhamir). The majority of Muslim will never touch alcohol in their whole life, and many will never have even seen it.

Postal Address: ***P.O Box 11639***
Jeddah 21463
Saudi Arabia

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Glimpses of Research in Medicine, in the Valley for the Last Two Decades.

Mohd. Yousuf*

JK Practitioner 1996; 3:4 260-262

More than twenty years of Medical research in the department of Medicine, Govt. Medical College, Srinagar is a matter of great satisfaction. The milestones of formal medical research starts in the career of medical student in the form of writing a thesis based on scientific plan, material and method and it is a part of pre-requisite for M.D. degree. P.G. course in Govt. Medical College, Srinagar started in 1966-67 and it saw a batch of very dedicated and talented students which among other included Dr. G.N. Qadri; Dr. Hanjura and Dr. Thusoo. I got a chance to work with them and continue to be inspired by their outstanding zeal for work to perfection.

Post-graduate medical research has been supervised by a galaxy of outstanding teachers like Lt. Col. S. Koul: Prof. A.M. Jan; Prof. S.N. Ahmad Shah; Prof. Zahoor Ahmad; Prof. G.Q. Allaqaband and Prof. S.N. Dhar and others who also headed various research programmes in other institutions like S.K.I.M.S.; Jammu Medical College and even abroad. Most of the research has been basic and locally oriented and whole of it may not find a place in foreign journals, but it is quite relevant to local conditions. So far about 170 scholars in quarter century have contributed to this treasure of scientific knowledge and fact finding. Research work has been broad based and encompresses almost all important systems like cardiovascular, respiratory, excretory, gastrointestinal,haematology, endocrine and nervous system. An abbreviated record of the research and the names of the chief investigators involved with it, is given as under:-

Cardiovascular System

Electrocardiography:-

- ECG changes in normal Kashmiris
- Stress testing in normal Kashmiris over the age of 30 years
- ECG study of arrhythmias complicating myocardial infarction
- Role of ECG in detecting effects of electrolyte abnormalities.

Hanjura (1967); Khan (1979); Gupta (1980); Rakesh (1983)

Arrhythmias:-

- Study of various arrhythmias in Kashmiri patients
- Clinical profile of patients with implanted permanent pacemaker
- Carbamazepine as an antiarrhythmie agent

Gandotra (1974); G. Ali (1986); Samia (1989).

Rheumatic Heart Disease:-

- Infective endocarditis as an important complication of RHD
- Prevalence of RHD in school going children of Srinagar city.

Sheikh (1980); S. Bhat (1983)

Ischemic Heart Disease:-

- Correlation of serum uric acid and cholesterol concentration in IHD
- CAD in Kashmiri population with special reference to serum Mg^{2+} and cholesterol
- Role of treadmil exercise test in evaluating patients with chest pain with normal ECG in IHD.

Jalal-u-Din (1976); Gupta 1978; Mattoo (1979); Sheikh (1987)

Myocardial Infarction:-

- Familial hyperlipidemia in case of M.I
- Precipitating factors in acute M.I
- Acute transmural M.I. in Kashmiri patients.

Kamili (1980); Kapoor (1985); Matta (1987).

- Serum Mg^{2+} and calcium estimation in M.I

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* *Prof. and Head*
Department of Medicine
Govt. Medical College Srinagar

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- Platelet adhesiveness, plasma fibrinogen and effect of sulpinpyrazone of platelet adhesiveness.
  Kamgar, Gul Bano (1981); A. Peerzada (1983); G. Mohd (1984).

Cardiomyopathy:-

- Puerpural cardiomyopathy in Kashmir
  Jalali (1985).

Hypertension:-

- Prevalence its correlation to serum Ca^{2+}
- Effect of high and low Na^+ intake
- Effect of diuretic and beta adrenergic blocking agents
- Correlation with glucose tolerance, serum insulin levels, lipid profile and left ventricular mass.
  Gupta (1983); Lone (1985); Sharma (1985); N. Ahmad (1986); Yousif (1987); Reshi (1993).

Pulmonary Tuberculosis:-

- Various aspects of clinical picture
- Relation with serum immunoglobulin levels
- ECG changes
- Drug toxicity.
  Hajini (1977); Shamasuddin (1977); Thussu (1978); Kachroo (1989); Qureshi (1991); Mubarik (1993).
- Various aspects of bronchial asthma.
  Wani (1987); Muzaffar (1991); Anjum (1991); Maqbool (1991).
- Environmental lung disease
  Saini (1979); Wandroo (1984)
- Pneumonias, chronic bronchitis, interstitial lung disease neoplasms, pleural disease.
  Nafee (1972); Nath (1974); Khan (1974); Lone (1977); Wali (1979); Saproo (1980); Koul (1984); Kotwal (1984).

Excretory System:-

- Acute and chronic renal failure
- Major glomerulopathies
- Polynephritis, UTI in children.
  Khan (1974); Bhat (1978); Shah (1980); Gupta (1983); Ahmad (1983).

Gastrointestinal System:-

- Bacteriological and clinical features of acute diarrhoeas, colonic diarrhoeas
- Protozoal and helminthic infestations in Kashmir
- Ascariasis in Kashmir with reference to diverse clinical presentation
- Hematemesis, melana, hematochezia
- Evaluation of gastric and output in Kashmiri patients in various gastric disorders
- Clinical and endoscopic profile of DU patients
- Role of upper GI endoscopy in UGI bleeding
- Effect of red chilly powder in normal and peptic ulcer patients
- Social aspects of DU
- Transit time in Kashmiris
- Lactose intolerance in DU patients
- Neoplasms of oesophagus and stomach in relation to dietary habbits, smoking and snuff.
  Khuroo (1971); Wani (1971); Ismail (1973); Afzal (1974); Lone (1974); Kotwal (1976); Malik (1976); Tagoo (1976); Zaki (1977); Beig (1977); Ahmad (1978); Yaseen (1979); Wafai (1979); Tickoo (1981); Badyal (1983); Thusoo (1984); Raina (1987).

Hepato-Biliary Disease:-

- Prevalence of hepatitis B antigen in healthy individuals of Kashmir
- Australian antigen in acute and chronic liver disease- Hazards of medical persons with reference to hepatitis B
- Drug induced hepatitis
- Cirrhosis of liver
- Ascitis.
  Bhan (1974); Malik (1974); Dewani (1974); Bhat (1978); Dar (1978); Ahangar (1979); Narboo (1979); Jhandoo (1983).

Hematology:-

- Platelet disorders with particular reference to hypertension and ischemic strokes
- Feasibility and safety of autologons transfusions
- Incidence of iron deficiency anemia in rural population.
  Bakshi 1974; Koul 1984; Rather 1984.

Metabolic Disorders:-

- Lead absorption in paper machie workers
- Prevalence of diabetes mellitus
- Osteomalasia due to anticarvulsaul drugs.
  Yousif (1971); Razdan (1977); Wahid (1977).

Central Nervous System:-

- Cerebovascular disease with particular reference to etiology, clinical picture CSF and CT findings
- Prognostic factors in sunarachnoid hemorrhage
- Clinical presentation of meningitis in Kashmir.
  Haroon (1973); Kudyar (1974); Singh (1977); Kanjwal (1983); Sirwal (1983); Shah (1991).

Conclusion

In conclusion it is gratifying to remark that we are working to raise our standard of research, focus on adequate funding, making resources available, raising standard of the supervision on scientific basis and speeding up our role in modern research in medical field.

I am sure that continued zeal of research scholars, laboratory workers and administrative staff, dedication of faculty staff, of department of Medicine and above all the patronage of University of Kashmir, our standard of research is going to be progressive, well applied and of high scientific standard comparable to any institution of the world as is evident from some of the latest research work especially on H. pylori and Ramzan fasting and peptic ulcer (Malik, et al) have found notable place in some international journals.

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"Magnetic Resonance Imaging & Proptosis"

Bashir Ahmad*; A.R. Nasti;**
Tejit Singh*; Sheikh Sajad******

JK Practitioner 1996; 3:4 263-264

The rapid proliferation of Magnetic Resonance Imaging technology is similar to that experienced by computerised tomography in 1970's. As an initial comparison, MR requires no ionising radiation to produce an image whereas the CT exposes the patient to relatively high doses of radiation. There is no definite proof yet that strong magnetic fields used in MRI are harmful.

The greatest advantage that MRI offers relative to CT, is the potential that it holds for tissue characterisation. The information obtained by CT is at the gross anatomic level. CT can detect differences only in tissue X-ray attenuation. MRI on the other hand holds the potential for characterisation of tissues at moleculer level. A variety of electromegnetic interactions within the tissue can be distinguished by MR. By varying pules sequences, magnetic fields and detection modes, it may be possible to image a variety of separate molecules and atoms.

Although CT and ultrasonography (USG) have been of tremendous help in the past decade or so, in delineating orbital space occupying lesions, yet they have their limitions e.g USG has it short comings in lesions located at the apex of the orbit and CT is relatively non-specific for tissue characterisation and degree of encapsulation. MRI is proving more useful and possibly superior to CT in the detection and delineation of orbital pathology. It is being used as a diagnostic modality for orbital lesions.

Physical Principles of Nuclear Magnetic Resonance:

Magnetic resonance imaging discusses the phenomenon whereby the nuclei of certain atoms when placed in a magnetic field can absorb and emit radio-frequency. The spectrum of absorbed or emitted radio-frequency depends upon the nucleus under observation and its chemical environment. Nuclei suitable for MR imaging are those which have an odd number of protons and neutrons. Hydrogen is the most commonly used nucleus. Although it is possible to image, by MR a number of different atoms including Hydrogen. Sodium, potassium and Fluorine, yet in actual clinical practice MRI involves only hydrogen protons. It is most abundant in biological tissues and thus produces the strongest emitted signal.

Nuclei have inherent spin, which is due to spins associated with the individual component protons and neutrons. When charged particles spin or move in any way, a magnetic field is generated around them, thus a proton can be regarded as a small freely suspended bar magnet spinning rapidly about its magnetic axis.

The term resonance is used to describe any physical system that oscillates between high and low energy status. MR thus involves hydrogen protons absorbing and releasing energy in the electromagnetic spectrum by transition between quantised energy levels. Such oscillations occur at very specific frequencies unique to each system. The process of MR signal generation can be thought of in two steps viz, stimulation to resonance and relaxation from resonance.

Stimulation to Resonance :

In a sample of tissue away from external magnetc field the tiny magnets are randomly oriented. When this tissue is placed in a strong magnetic field the tiny proton magnetic movements will tend to align with the field producing a small field of their own. This small induced field in the tissue is called the magnetisation vector M. When a second oscillating magnetic field of resonance frequency matching the Larmor frequency of hydrogen proton is applied "magnetic Resonance" occurs and the magnetisation vector rotates away from its original alignment. This absorption and remission of electromagnetic energy is the process of nuclear magentic resonance.

Relaxation from Resonance:

The process by which stimulated protons release their energy and return to original alignment is known as relaxation. Several things may happen to the energy that is released:

a/ It may be released as radio-waves and recorded as MR signal, using RF coils as an antenna receiver.

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*Professor and Head, **Associate Professor,
***Assistant Professor, ****Registrar
*****Sr. Resident Department of Ophthalmology
Government Medical College Srinagar.*

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b/ It may be lost irreversibly to the surrounding tissue "lattice in the form of heat or induced molecular motion (TI relaxation, longitudnal relaxation, thermal relaxation or spin lattice relaxation).

c/ It may be transferred reversibly to other protons undergoing resonance although phase uniformity may be lost (T2 relaxation, transfers relaxation or spin-spin relaxation).

The process begins when the stimulating resonance frequency coil is turned off. The obliquely oriented M. vector may decompose into two components M1 and M2 parelled and perpendicular respectively to the transverse component. These two relaxation processes are known as T1 and T2 relaxation respectively defined to above.

In general the value of T2 is more closely related to tissue state (solid or liquid) than T1. The tightly held and closely placed hydrogen nuclei in solids are more efficient at spin - spin interaction than are those in loosely structured liquids - Accordingly, dense solid tisues with large protons have the shortest value of T2- Waterly tissues and fields like CSF have very long value of T2.

M.R. signal strength/Image contrast:

Four physical characteristics of a tissue being imaged will determine the signal intensity being detected by the imager and encoded in grey scale:

They are :

a/ Hydrogen density
b/ Motion of hydrogen protons.
c/ T1 relaxation time
d/ T2 relaxation time

However the manner in which information is gathered can be varied to influence the relative contribution of each of these parameters to the signal. Since multiple tissue variables are expressed in a given images a given choice of instrument parameters may produce a casual similarity of signal intensities from disparetic tissues, making lesions to be indistinguishable from their surrounding tissues.

To get over this, the ability to vary the imaging sequences to highlight the different combination of T1 and T2 relaxation, characteristics is crucial. For a spin echo type imaging, a long T1 relaxation time or short T2 relaxation time will tend to decrease relative signal intensity whereas a short T1 or long T2 relaxation time will tend to increase signal intensity.

The pulse internal (TR) is the time between repetive RF perturbutions. The echo delay (TE) is the time elapsed between RF perturbutions and the point in time when the signal (echo) emitted by the protons within the tissue is sampled. Both TR and TE are parameters under computer control. A short TR and a short TE (TR 0.5 seconds; TE 28 msec) maximise the relative T2 contribution to signal intensity. They are referred to as T2 weighted images. Images can be routinely obtained in axial, coronal and sagital planes. The section thickness of the images depends on the matrix used. A matrix of 128 x 128 (standard) 0r 256 x 256 (high resolution) pixels can be used with a special resolution of 1.7 mm for standard images and 0.8 mm for high resolution.

MR Scanner System:

The MR scanner system essentially consists of:

a/ Superconductive magnet of usually 0.5 to 2.5 tesla strength housed in a large cylindrical frame with accessory magnets to establish and regulate the powerful magnetic fields required for magnetic resonance to take place. The room containing the magnet is specially designed and shielded lest the fringe magnetic fields produced by the magnetic interfere with nearby electronic equipments or extraneous electro-magnetic radiation interfere with the production and detection of small MR from with the body.

b/ Gautry on which the patient lies and that can be moved into the centre of the magnet.

c/ Radio frequency coil placed close to the patient to stimulate and record MR signal. Different sizes and shapes of RFcoils are used depending on what part of the body is being scanned; head and neck coil, body coil and surface coil.

d/ Devices such as operator's console & separate physicians console for reviewing of images.

e/ Room to house peripheral devices as computer, disc driver, amplifiers, voltage stablisation etc.

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Ultrasound Pictures of GIT Ascariasis in Kashmir

JK Practitioner 1996; 3:4 265

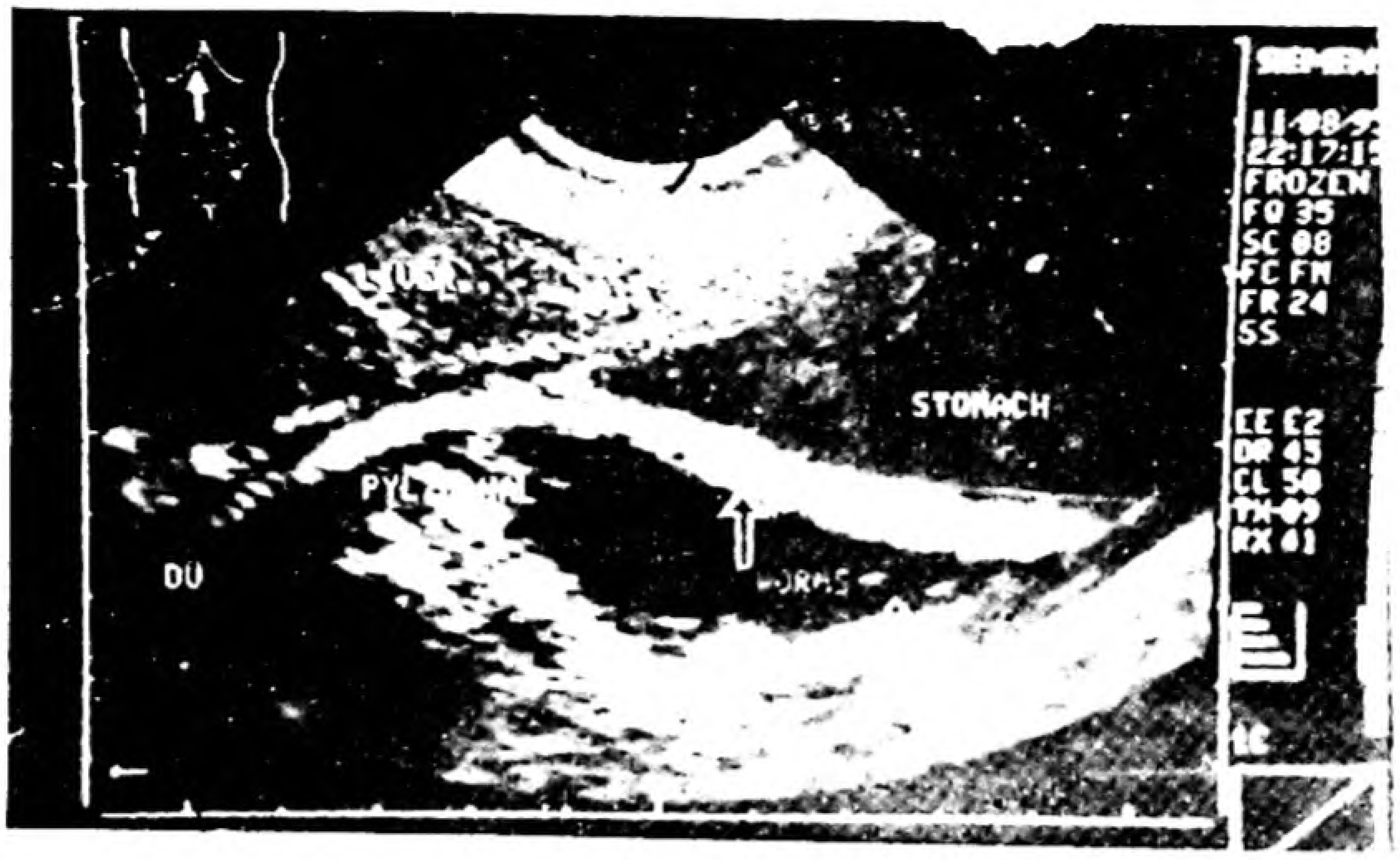
FROZEN
FQ 35
SC 08
FC FN
FR 24
SS
STOMACH
EE E2
DR 45
CL 50
RX 41
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Fig. I. Gastric Ascariasis (shown with arrow)

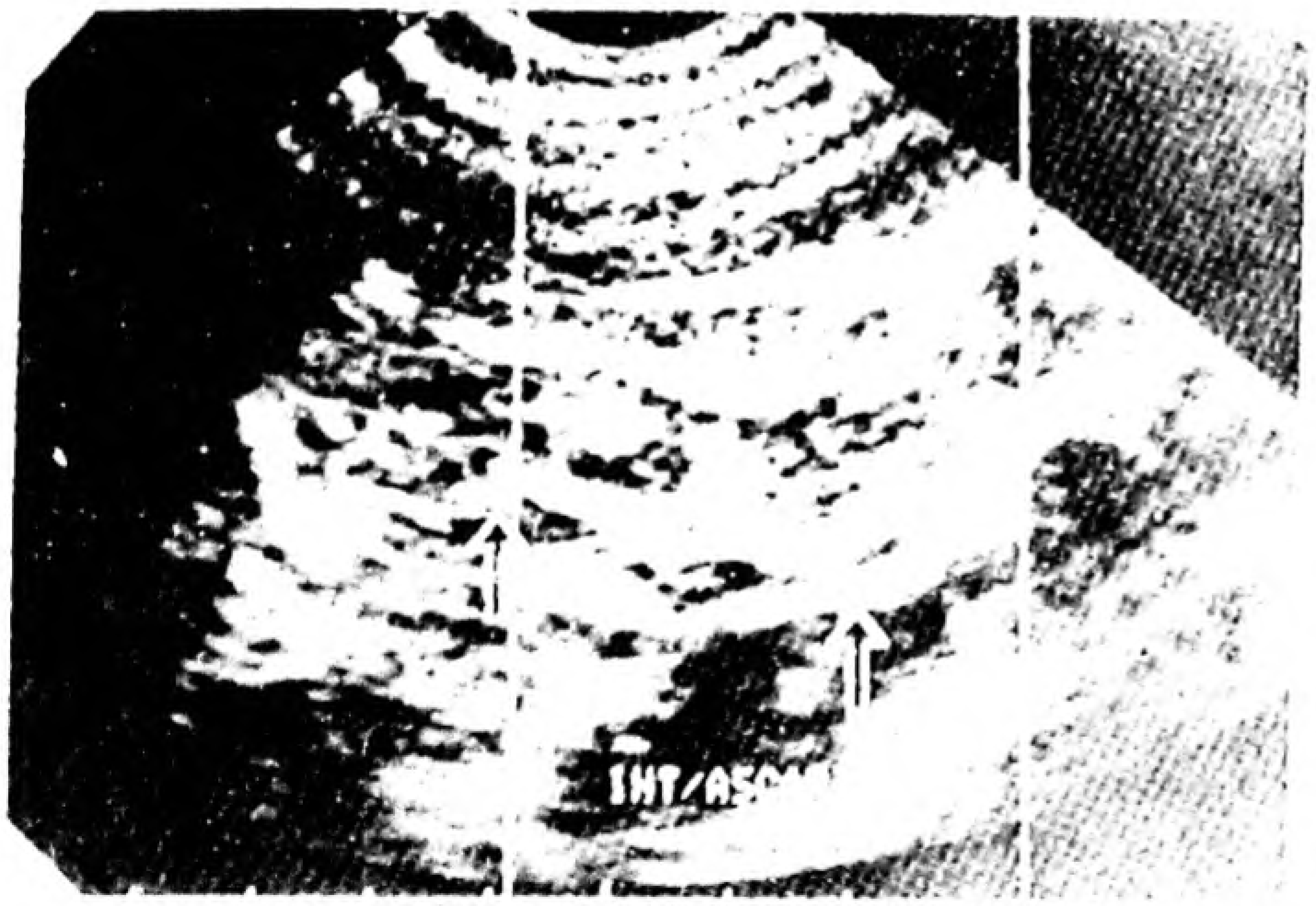

Fig. II. Intestinal Ascariasis (arrow)

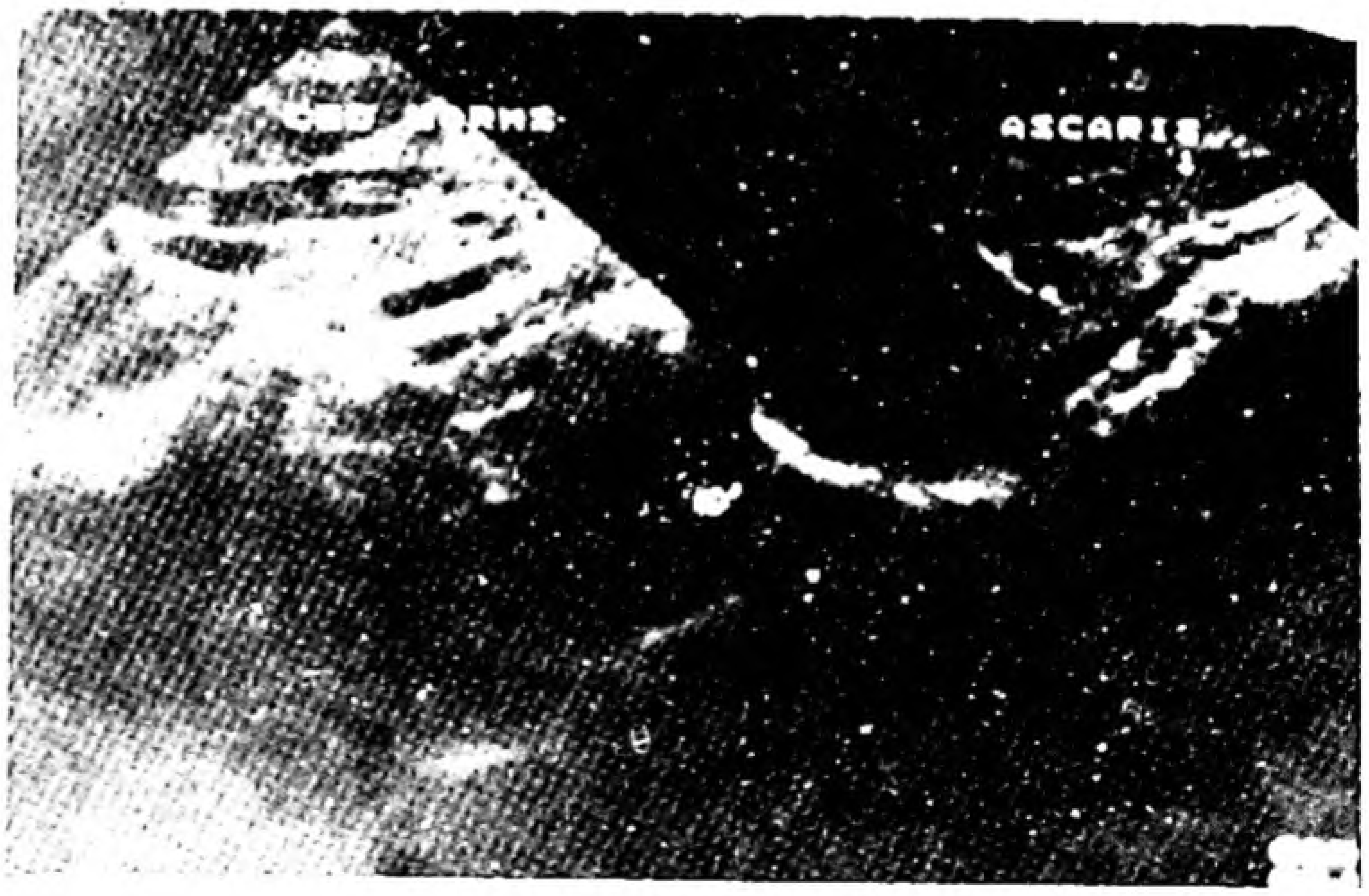

Fig. III Biliary Ascariasis (arrow)

Authors:
G.M. Malik MD. FACG*; S. Kadla**
* Associate Professor
Head GIT Division Deptt. of Medicine
Govt. Medical College Srinagar P.O. Box 884
** Registrar Deptt. of Medicine
Govt. Medical College Srinagar (Kmr.) 190 010

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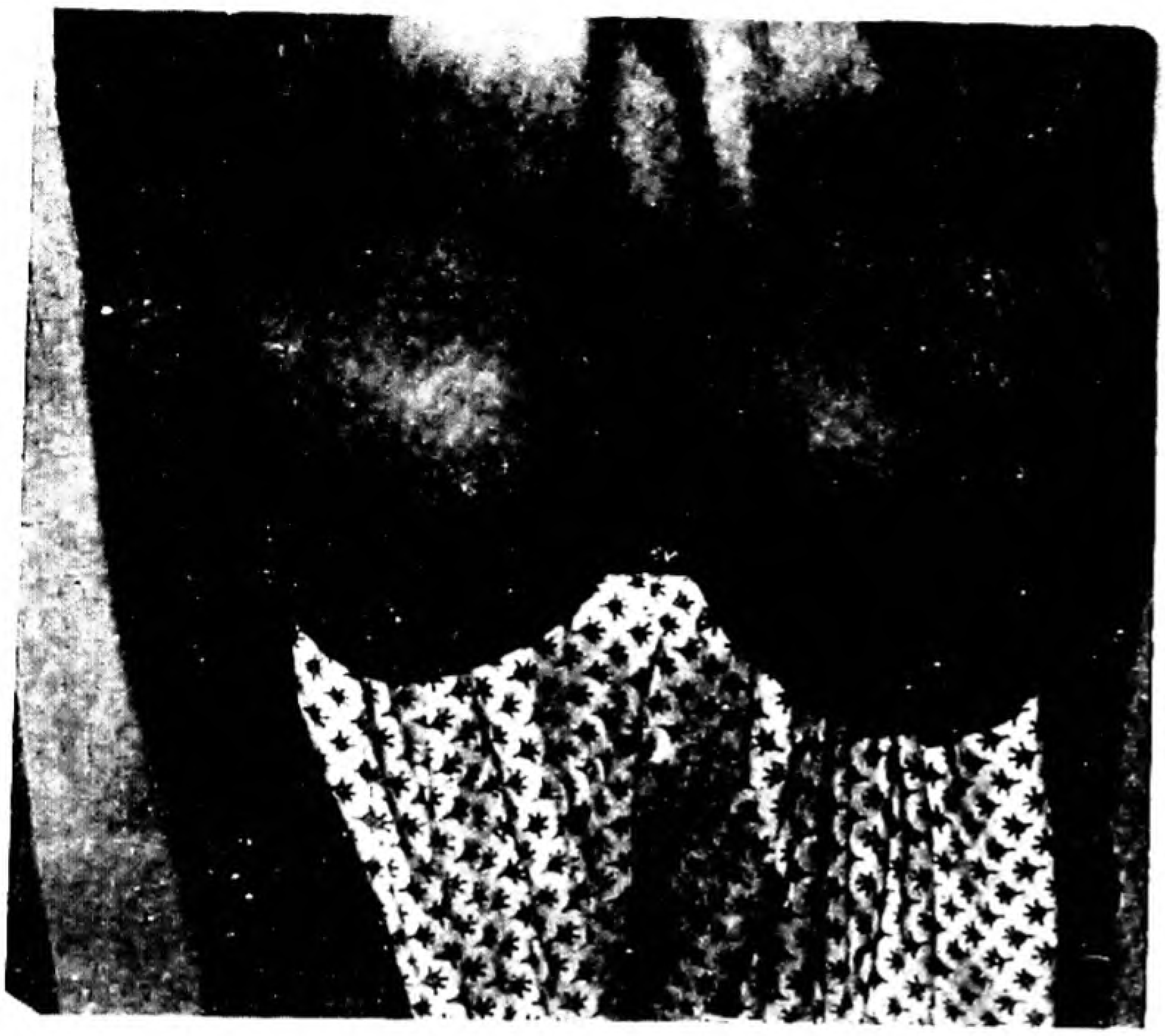

Fig. I

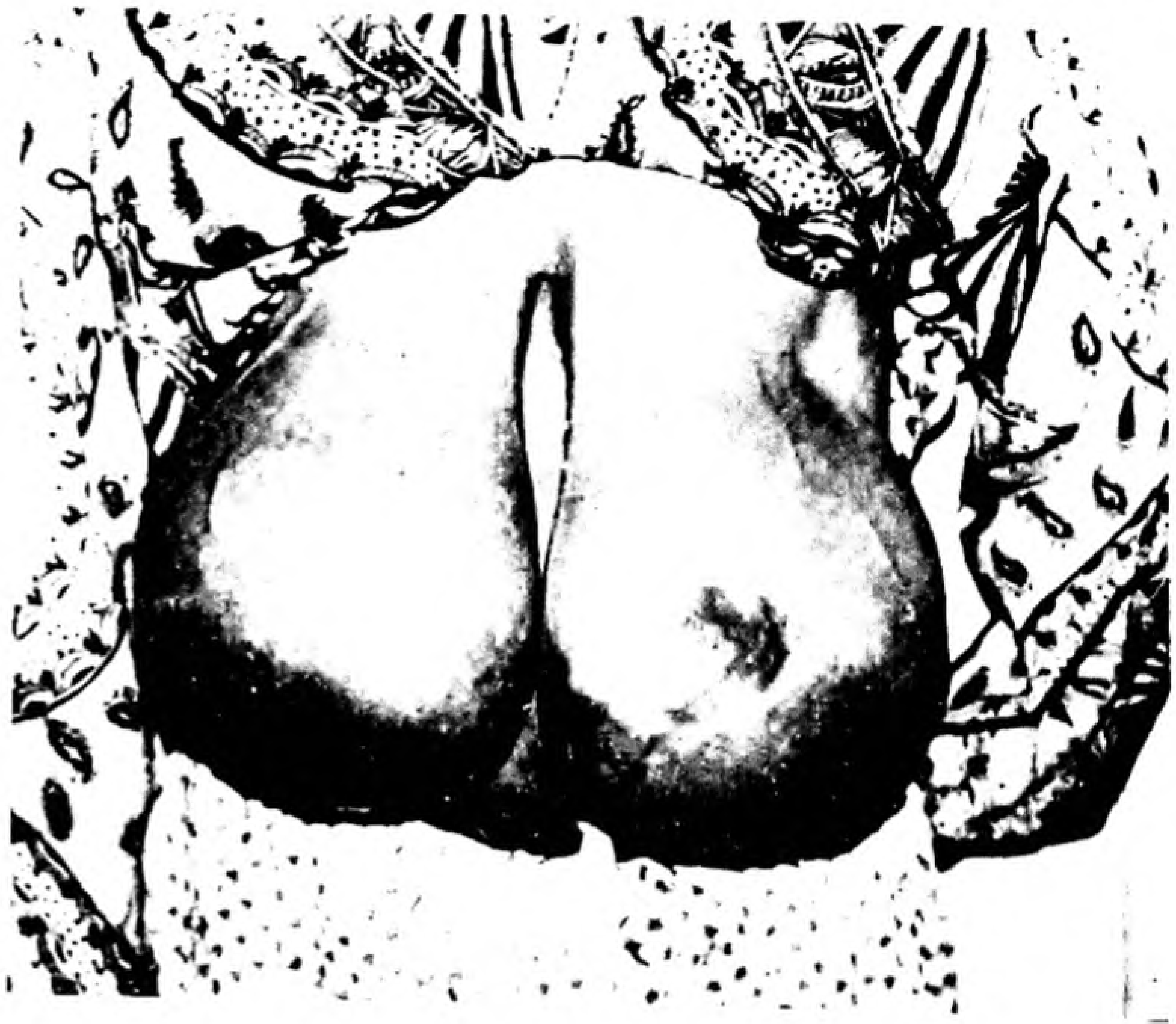

Fig. II

Case Summary

A 13 year old girl was referred because of progressive, painful enlargement of both breasts, starting six months back and becoming massive within few weeks. Examination revealed a 145 cm tall, 36 kg girl, in whom weight bearing of the breasts itself was uncomfortable. Breasts were large, erythematous with multiple ulcerations and cresting of the overlying stretched skin (Fig. I). Gynecological assessment was normal except she had not achieved menarche. Hormone profile revealed normal T3, T4, TSH, LH, FSH, growth hormone, prolacting and cortisol. A course of danazol, 200mg administered orally twice a day was instituted besides supportive measures and antibiotics with this ulcerations healed and breast volume (measured by immersing both breasts in water and nothing the volume of water displaced) decreased from 4700 ml to 3100 ml after six months (Fig. II) Thereafter, she started getting side effects of danazol therapy in the form of alopecia. Danazol was stopped and she was subjected to surgery (bilateral reduction mamoplasty). There has been no recurrence for 6 months of following

Diagnosis: PUBERTY GIGANTOMASTIA

Authors: A.H. Zargar, D.M; S.R. Masoodi, M.D; B.A. Laway, M.D;S Iqbal, M.Cg, Departments of Endocrinology and Plastic Surgery, SKIMS, Srinagar

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MEDICAL ETHICS: Challenges and Response

Syed A. Tabish*

JK Practitioner 1996; 3:4 267-272

Historical, demographic and scientific changes occurring in twentieth century have played a key role in the development of medical ethics as a discipline. Health care professionals have seen parallel changes in the nature and goals of medical practice, and professional organisations have attempted to codify the profession's response to these challenges.

Human endeavours to improve the quality of health care has been documented as early as 2000 BC. In classical Greece, Hippocrates required his students to swear to an oath that the care administered to patients by them would always be to the best of their ability. By the time of Renaissance, a commonly practiced "oath of physician" in Europe pledged that physicians would " Prescribe regimens for the good of my patients according to my ability and my judgment and never do harm to anyone _______". From these humble through well intentioned beginning have come present understanding and standards governing the implementation of quality Assurance activities.

Many influences have the potential of leading to the impersonalisation of medicalcare:

- Vigorous efforts to reduce the escalating cost of health care:
- The increasing reliance on technological advances and computerisation for many aspects of diagnosis and treatment:
- The increased geographic mobility of both patients and physicians.
- The growing number of closed system' arrangements, such as health maintenance organisations, in which the patient has little if any choice in selecting a physician;
- The need for more than a single physician to be involved in the care of most patients who are seriously ill; and
- an increasing reliance by patients on legal means to express their disappointment with the health care system (i.e., by malpractice litigation).

Given these changes in the health care system, maintaining the human aspects of medical care and the empathetic qualities of the physician is a particular challenge.

Patient care begins with the development of a personal relationship between the patient and the physician. In the absence of a sense of trust and confidence on the part of the patient, the effectiveness of most therapeutic measures is diminished.

There have been increasing demands for physicians to account for their actions (the way in which they practice medicine) by meeting certain standards prescribed by various governments and voluntary professional organisations. The hospitalisation of patients whose healthcare is reimbursed by the government and other third parties is subjected to utilisation review (the physician must defend the cause for and duration of a patients's hospitalisation if it falls outside certain average standards).

Medical Ethics

Medical ethics involves the study of general problems relating to healthcare, health care institutions, and biomedical research. The ethical debates currently in medicine have arisen from new technologies, increased respect for patient's rights, and financial restraints. Interest in medical ethics will remain strong as long as ethics addresses the concrete concerns of patients and practitioners.

Virtually every contact between patient and a health professional contains moral dimensions, especially those contacts that place demands on the health professional. Most often the values of the patient, the health care worker, and the sponsoring institution conform, and there is no moral conflict. However, when values conflict, there is need for medical ethical reasoning.

The hospital has a responsibility to protect its patients and staff when it knows or should know that malpractice was likely to be committed upon them. The health care organisation exercises its corporate accountability through its governing board. The board delegates its responsibilities of patient care monitoring and assessment to management and the medical staff, and looks to the Chief executive officer and the organised medical staff to conduct Quality Assurance activities. The moral commitment by the members of the governing board to pursue patient's interests can only be accomplished by

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***Administrator*
Medical Institute Soura Srinagar Kashmir 190004

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a concerted effort of all, concerned.

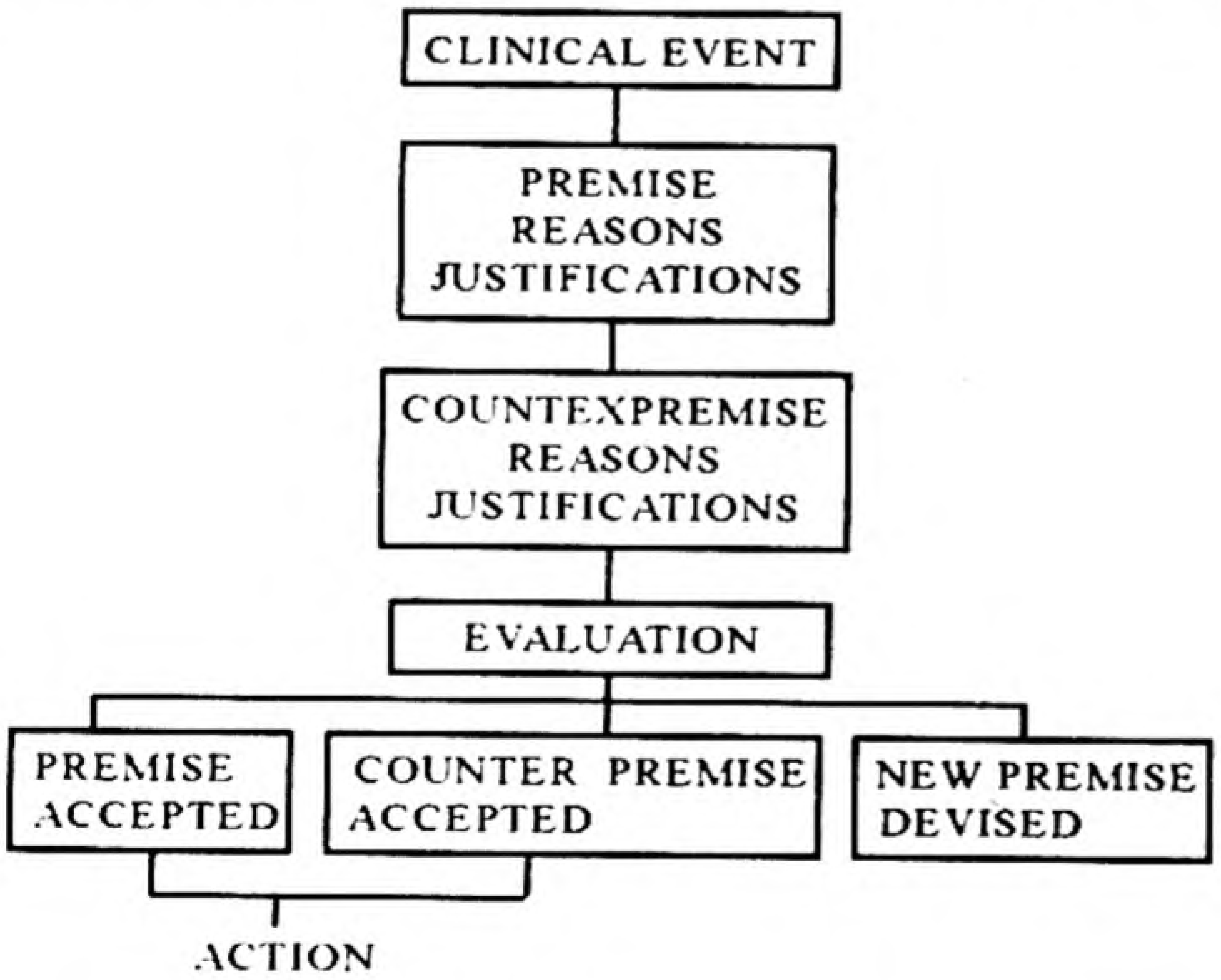
Ethical Decision Making

Applied medical ethics seeks to relate general normative ethical theory to concrete decisions that must be made about specific situations. Standard decision-making procedures can be used (Fig.1)

Elements of the case method

- -Premise: The premise is an action statement regarding conduct that should be initiated or discontinued because of moral obligation of the physician to the patient or to an involved institution.
- -Ethical Argument: It consists of reasons for the precise and justifications for the reasons.
- -Counterpremises: For each premise, there will be one or more counterpremises.
- -Evaluation: Consists of weighing the several reasons to determine their relative importance and to select the most important elements.
- -Action: The ethical decision making is directed to an action or to a determination not to act. Eventually one may need to proceed without the agreement of other interested parties

Ethical Decision Making in the clinical setting



Principles of Medical Ethics

Autonomy

Among the broad Principles that help determine the characteristics of a right or wrong moral action is the principle of autonomy, manifested as respect for persons and their free choices. Every human being has a right to determine what shall be done to his own body. A physician's respect for the persons who are his patients derives from respect for human life itself, and supports the individual patient's right to determine her own life course, within the limits of avoiding harm to others. The priniciple would direct that physicians act so as to respect the values of the patient, even if, and especially when, there is a possibility of harm to the patient in doing so.

1. Truth - telling. The recognition of autonomy required that the patient have access to the truth about his condition.
2. Decision making - one marked of autonomy is the ability to make rational decisions, especially when considering one's own future. Truly moral decisions incorporate respect for the patient's own desires and needs.
3. Respect for individuals. The illness often weakens a patient's independence: it is, therefore necessary to help the patient according to his individual needs so that autonomy is practiced.

Thus, a patient's uncoerced and competent decision cannot be overridden by a physician's appeal to patient best-interest.

Respect for individual autonomy include:

- the obligation to disclose information.
- to participate in the ongoing process of obtaining informed consent for treatment from patients.
- to assist the patients in choosing the least restrictive alternative when decisions adversely affecting autonomy are faced.
- ensuring privacy and quiet in the physician - patient encounter
- allowing adequate time for discussion and kind treatment, and
- remembering that each person, no matter how debilitated, is a member of the human community and thus deserving of fundamental dignity and respect.

Beneficence and Paternalism

The principle of beneficence is concerned with the duty to help others by doing good for them and helping them to avoid harm. The benefits and goals of the healthcare, based on the physician's scientific proficiency and experience, include prevention, care, or amelioration of disease, disability, or pain and suffering. Beneficence includes all efforts to increase the health of the community.

With a competent and autonomous patient, agreement on the goals of health care is usually present and the physician is free to act out of beneficence and in the mutually agreed upon best interest of the patient. In persons of diminished capacity, in contrast, it is often necessary to act solely on the basis of patient best-interest, without a patient's autonomous and informed consent.

Paternalism is the reliance of healthcare professionals on their own judgment to determine a patient's best interest. Paternalism is viewed as a social safety net that rational persons would choose to have beneath them in case of future loss of mental capacity. Paternalism may be justified under carefully defined circumstances.

- if the harm avoided or benefit gained outweigh the loss of self-determination resulting from the intervention
- if the patient is too sick to choose freely, and
- if other person in similar circumstances would be likely to make the same decision.

Fidelity

The principle of trust is embodied in the fiduciary relationship of physician and patient. This principle requires the physician to act in good faith towards the patient, balancing the respect for the patient self-determination with concern for patient welfare, and clearly placing the interest of the individual patient above all other considerations. Manifestions of fidelty include:

- the preservation of confidentiality, and
- and obligation requiring increasingly strenuous effort in an era of computerized medical reports and large multidisciplinary teams of health providers.

Fidelity is the duty to observe the pledges made by the profession.

1. Truthfulness. Telling patient the truth within the bounds of fidelity
2. Confidentiality. Access to a patient's record may be obtained legally by health professionals, hospital personnel, and insurance or government representatives. The guidelines include;

- limiting access to those who have a legitimate need
- using pseudonyms and altering other identifying data when presenting cases in conferences and teaching situations.

Justice

It refers to fairness or equitable access to treatment. Equitable distribution of benefits and burdens constitute the subject matter of the principle of justice. The physician must balance the interests of his patient with these of hospital, insurers, society and physicians own practice.

- Procedural justice. One means of ~~just~~ distribution is the establishment of rules. To be fair, exceptions to rules should be clearly built in the procedures. The distribution of services on the basis of "first come, first served" is an example.
- Distributive justice. Equal sharing may not always be the most just way to distribute benefits or burdens, additional claims may be involved. Claims requiring decisions based on distributive justice arise when resources are limited, thus, reduction on spending of healthcare generate the questions of who shall have the healthcare and who shall not.
- Compensatory justice. Punishment as retribution is one form of compensatory justice. The more positive aspect is the attempt to reward victims for losses that are not the consequences of their own action.
- Individual and Society. Decisions made for the "good of society" are particularly painful when they discriminate against an individual patient. The physician is often caught between the primary duty to the patient and obligations to society.

The priniciple of justice requires that equity of access to care be sough in situation of scarcity.

Nonmaleficence

The duty to do no harm is one of the oldest guiding principles of the medical profession.

- Deliberate acts to harm. Only under limited concepts of self-defense is it permissible to harm another person. The exception in medicine is a harm (hurt) needed to help, heal, or cure the patient (e.g. surgical incision).
- Calculated risk. When possible harm may occur, there must be a compensating and compelling possibility of benefits to justify the action. The side effects of medications are calculated risks when the benefits are most important (e.g., to use almost toxic doses with certain tumours). For health professionals, standards of "due care" include knowledge, skills, and perseverance.

Age Limits

Several ethicists have proposed an arbitrary age limit beyond which entitlements for certain types of medical care would be denied (Callahan D. 1990). It is argued that the recent success of medical technology has created the unprecedented public expectation that the inevitable physical decline of old age and death has been lost in a futile effort to regain youth. Further the medical use fullness standard for decision-making is described as unrealistic in the face of advances in life - extending technologies (such as heart transplant). The medical usefulness standard is based on central moral principles of compassion and beneficence, departure from this standard may put the ethical standing of the medical profession at serious risk.

It is being argued that the medical usefulness criterion (individual assessment of prognosis and likelihood of benefit from a given intervention is a sturdier and fairer basis for allocation of medical resources (Levinsky NG, 1990) .

Moral Problems

1. Limiting Technology

Technological advances and knowledge explosion (sophisticated diagnostic and therapeutic techniques) allow physicians access to procedures that alter the traditional relations between physician and patient.

- Initiating life supporting treatment, legal and moral uncertainties surrounding the removal of life-support systems have in turn, raised issues about initiating treatment at the onset of a medical cirsis.
- With holding life support techniques has been better accepted than not initiating treatment, yet both decisions are difficult.
- Terminating life. Physicians are debating whether terminally ill patients be assisted to die 'by active means'.
- Control and autonomy. Many of the new technologies promise methods of mind and behaviour control. Which have beneficial effects for some patients but which could also be used by repressive governments, or even by paternalistic health professionals.

2. Limiting Resources

The availability and type of medical care are restricted by the choices of individual physicians about their practice and through the process of governmental budget settings. Choices of microallocation (decisions affecting individuals) and macro allocations (decisions about which goods are available) produce many ethical questions - the right to treatment, individual Physician's decisions, and medicine and other social needs.

3. Limiting Research

The new technologies are the result of the burgeoning research that occured after the Second world war. The issues are:

- Deception: It should be determined when, if ever, deception is necessary in research.
- Informed consent: The rights of individuals to choose risks has led to the move for informed consent in research.
- Privacy: The need to maintain the confidentiality is a constraint on researchers.
- Reporting unethical research.

4. Limiting risk of disease

The perpetual problem of the medical profession to weigh the risks of the increased incidence of a disease against other public or private goods, such as the invasion of the Privacy, the rise of the unncessary panic, and confidence in healthcare has been illuminated by the health problems generated by AIDS DISEASE. The ethical concerns include - curing for infected individuals, reducing the spread of the disease, finding treatment and eventually cures, and accepting AIDS as a professional risk.

Responding to Challenge

The determination of decisional capacity is a clinical process involving patients, health professionals and family, which is decision specific: that is, it will vary depending upon the nature and the consequences of decision to be made. Assessment of capacity for various levels of decision-making occurs in the routine interaction of patient and physician. Efforts to identify and reserve possible contributors to diminished capacity such a drug toxicity or reversible illness, are critical to the promotion of patient autonomy in medical decisions.

Informed consent requires open and comprehensible communication between physician and patient regarding the nature, risks, and benefits of the various treatment options, discussed, and understood in the context of patient's values, goals, and life preferences. The requirements of informed consent are:

- adequate disclosure of information by the physician to the patient;
- patient capacity to understand that information and to utilise it to make a decision based on their own values, and
- a voluntary or non-coerced decision for or against the offered intervention by the patient.

The disclosure of full information in the consent process is usually deemed adequate if it meets either a community standard of practice or reasonable person standard. The reasonable-person standard is preferred for its intention to provide information that a reasonable person would need or choose to have in making such a decision. The community's standard of practice, conversely, may not adequately promote patient autonomy. Careful effort on the part of physician to determine and acknowledge patient's concerns and interests often leads to greater mutual understanding and an acceptable compromise.

In case where a patient lacks decisional capacity, informed consent cannot be obtained and other persons must be brought into the process. Whoever is closest to the patient should be asked to represent his/her wishes as a surrogate decision maker, and to use this information in the context of the physician's recommendation to arrive at a decision. In true medical emergencies , treatment can be initiated without a formal consent process.

While deciding to forego life sustaining therapy every effort should be made to provide only those treatments known to be effective and cost-efficient, but the physician should not place the gate keeper function ahead of obligations to individual patient care.

The decisions and wishes of patients regarding life-sustaining therapy should be respected in those who are able to understand and deliberate on the risks and benefits of the treatment and its alternatives. Even under circumstances where the physician cannot or will not accede to a patient's request, continued general medical care or, at a minimum, referral to another professional, is required.

When patients loose decisional capacity, other persons must decide for them. If the decisions of the surrogates are to reflect the patient's wishes, they must be based on the knowledge of the values, life styles, beliefs, specific requests (if any) of the patient as they apply to medical treatment. The Value History can also be considered. The objective of the value history is to enable decisions about care that are based on patients belief's and wishes that serve as reliable guides to health care professionals when patients are incapable of participating in the decision process. This history should be clearly recorded in the medical record for the future use. The process begins by the general identification of basic life values regarding preservation of a reasonable quality of life versus preservation of life at any cost. The history also seeks to define further and rank patient's values about dependency on others, burden on family members, and pain and suffering, among others.

Advance Directives permit prior expression of patient wishes for health care, to be used in the event of mental incapacity. The living will is an example and includes a statement of patient wishes for care in the event of terminal illness where recovery is unlikely. Patient can also designate a specific person as a proxy health care decision-marker in the event of his future incapacity. The advantage is flexibility for decisions in the wide variety of circumstances likely o arise at the end of life.

When medical decisions must to made for persons of diminished capacity, and as is usually the case, no advance directives or valve history are available, the decision-maker society closed must be made by a proxy decision-maker, usually a close family member or friend. The proxy should use the principle of substituted judgement in arriving at the decision. The substituted judgment standard requires that the proxy attempt to arrive at the decision that the patient would have, if she/he were still able to choose. The intended result is that the patient's definition of well being is respected through surrogate's effort to respect the patient's wishes, even in the face of diminished mental capacity. The substituted judgement standard requires reliable evidence of the patient's previously stated views and actions, which should be as specific and as relevant as possible to the current

To arrive at decisions for patients of diminished capacity, the Best interest Standard is widely used. The standard rests on protection of patient welfare as defined by broad social criteria. General criteria for defining patient best interest in medical decisions include concerns such as the relief of suffering, restoration of function or self-determination, length of life, and the quality of that life for the patient.

The presumption to resuscitate is based on the principle of 'erring on the side of life', recognizing that a delay in judgement regarding resuscitation has the irrevocable consequences of death. The principle of patient self-determination is accorded priority in determining resuscitation status. Competent patients should have the right to accept or refuse resuscitation measures, after having been fully informed of the benefits and risks of the procedure in their specific case. In persons of diminished capacity, the decision must be made by surrogates, utilising prior expressions of wishes if such are available, and the best interest standard if they are not. The undertaken, as far as possible, from the patient's point of view, utilising substituted judgement.

Euthanasia

Mercy killing (as distinguished from decisions to forge life sustaining therapy), is being widely debated. Some people believe that life is sacrosanct and its taking away cannot be justified on any grounds; that life begins at conception so even abortion is not permissible. But according to some moral philosophers only a person has a right to life (also a right to death). And only a self-conscious, rational, autonomous being is a person. A being lacking these qualities is a non-person and, therefore, has neither the right to life nor to death. The life and death decisions for non patient persons can be made by persons intimately associated with the non-persons. In this philosophy, abortion is permissible. It is also permissible to let die severely handicapped new borns, persons in irreversible coma or vegetative state, and demented aged. Euthanasia can be:

- Voluntary passive
- Voluntary active, and
- Involuntary passive

In passive Euthanasia, the treatment is withheld and the patient is allowed to die. In double-effect, the physician administers drugs which will relieve pain and suffering, but which, the physician knows, will also shorten the patient's life. In active euthanasia, the physician actually helps the patient die.

While passive and double-effect euthanasia are by and large acceptable to medical community, the active euthanasia is not. And herein lies the dilemma.

Compassionate and expert terminal care, including sophisticated pain management techniques, should obviate patient requests for euthanasia. If the dose of analgesic required to control pain also poses the risk of slightly shortening the life of a suffering terminally ill patient the medication may still be given since the objective and the intention of the treatment is the relief of pain and not death. The amount of such risk and its acceptability must be assessed in the context of the patient's life and values (Meier, 1983).

Research

Quality medical care and respect for the individual's right to contribute to society require that the elderly be permitted to participate in research: careful ethical analysis and protective guidelines should maximize the individual and soceital benefits of such research, and minimise its risks.

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1. PATHIN : Amoxycillin Trihydrate
500 mg Caps
250 mg Caps/Dis - Tab
125 mg Kid - Dis - Tab
125/5 ml Dry Syrup

2. TRAMIN : Analgesic, Anti-Inflammatory with muscle relaxant effects.

3. AVRIL TAB :
AVRIL SUSP. : Decongestant, Anti allergic and Anti pyretic

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Practitioners Column:

Migraine - New Horizons

A. Salam*

JK Practitioner 1996; 3:4 273-275

Summary

Migraine presents frequently in general practice. About 10% of the population is affected and over 3 million working days are lost each year as a result of migraine attacks. Reassurance and advice about trigger factors is as important as the selection of drug therapy in management of migraine. Prophylaxis should be prescribed to ensure that attacks are kept to minimum.

Introduction

Few medical conditions have been recognized as long as migraine. First described by A. Cappadocier in 2nd century A.D. Galen (130-200 A.D) called it hemicrania - (from Qreck-hemi kranios) and Roman translated the world to later hemicranoulong later corrupted to hemigranea and hence migranea, current name migraine. The French translation gained acceptance in 18th century and finally the term migraineur was used for the sufferer of this type of headache.

The Research Group on Migraine and headaches of WFN 1969 agreed on following definition of migraine `a familiar disorder chararcterised by attacks of headache widely variable in intensity frequency, and duration. Attacks are commonly unilateral and are usually associated with anorexia, nausea and vomiting. In some cases, they are preceded by or associated with neurologic or mood disturbances. All the above characteristics are not necessarily present in each attack or in each patient. Migraine is classified according to the scheme devised by the Headache classification committee of IHS 1988.

Pathophysiology

Despite the absence of an animal model, the lack of a reliable trigger factor and the difficulty of studying spontaneous attacks, several theories have been proposed:

i) Spreading oligemia (Olesen 1987) and spreading depression of Leao (1944) @ 3-5 mm/mint.
ii) The platelet theory (Hamington - 1986)
iii) Neural mechanism originating in the brainstem including the Locus Ceruleus (Lance et al 1985).
iv) Substance P and the trigeminovascular connections of the trigeminal nerve (Moskowitz 1984) and
v) Dystonomia involving the noradrenergic nervous system (Welch - 1987)

The evidence comes from investigations of migraine patients using rCBF, magnetoencephalography (MEH), magnetic resonance spectroscopy (MRS) and platelets. These are consistant with the hypothesis that migraine represents a state of central neuronal hyper-excitability (CNH). Low Mg+ in the cells is the gate-keeper of NMDA receptor and therefore rendering the nervous system more susceptible to Glutamatic-induced SCD. Also depressed EC Mg results in cellular extrusion of K+ in exchange for Na+, further more inducting or intensifying SCD. Finally low Mg open the non-NMDA dependent Ca+ channels resulting in IC ca+ accumulation, excitatory amino acid release and subsequently SCD.

In typical migraine sequence, the release of many substances precipitate a cascade of events that includes platelet aggregation, release of platelet serotonin and Tx, A2, activation of Postaglandins, production of kinin and localised sterile arthritis. The resultant symptomatic focal vasoconstriction is followed by reactive vasodilation, largely in the extracranial cerebral circulation, stretching pain sensitive arterioles and produces the familiar throbbing pain of migraine.

Clinical features

Migraine can begin at almost any age, most commonly, the initial attack occurs during the teenage years and by 40 years of age 90% of afflicted persons will have had these attacks. After puberty migraine is more common in females whereas in children there is a small preponderance of male subjects. A family history is present in upto 90% sufferers. The annual prevalence rate of migraine is estimated to be 20% to 25% (Watus-1978). Once migraine has developed, it tends to recur with varying frequency throughout much of a patients's life. Attacks have tendency to get milder and less frequent in later years.

***MD, DM**
Asstt. Prof. & Neurologist
Deptt. MedicineSKIMS, Srinagar, Kashmir - 190 001

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Different types

Common migraine:	Common, headache lasts for hours to 1-2 days associated with anorexia, nausea, vomiting, no prodrome or aura.
Classic migraine:	Vascular headache with sharply defined transient visual and other sensory or motor prodromes or both. Visual symptoms more common. Sensory symptoms include paresthesia or numbnes, headache usually unilateral
Complicated migraine:	Associated with neurological deficit for more than 24 hrs, after headache infarction rare, death reported.
Symptomatic migraine:	Secondary to an underlying structure cause such as AVM/cerebral tumor
Hemiplegic migraine:	A.D/hemiplegia which outlasts the headache.
Ophtalmoplatic migraine:	Headache associated with transient uniocular visual loss without overt ocular or carotid pathology
Basilar migraine:	Bilateral loss/disturbance of vision-associated with ataxia, vertigo, tinnitis etc, followed by occipital headache, commoner in adolescent girls.
Mig. Equitants:	Aura occurring in isolation without headache or systemic upset.
M. Coma:	Very rare, familial, associated with cerebral edema, hyperpyrexia etc.

Trigger factors

Stress. Depression. Fatigue, Exercise,
Head injury, Menstruation
Drug-oral contraceptive, vasodilators,
Changes in sleep pattern
Changes in weather-extreme cold/heat bright light-T.V. flickering Irregular meals/prolonged fasting
Alcohol
Diet: Cheese - chocolate. citrus fruits. fried foods, mushrooms, onion. strong tea or coffee, dairy products

Management

It is important that patients be made to feel that the treating physician/neurologist understands their complaint of headache and will not simply dismiss them as having headache for psychological reasons. A normal CT scan offers considerable reassurance.

Avoidance of trigger factors is important in the management of migraine.

Exercise programmes to promote well being, self confidence, correction of dietary excesses and avoidance of prolonged fasts and irregular sleeping habits can be helpful.

Long term stress management may require psychotherapy, biofeed back, relaxation training and hypnosis. Pain management programs are helpful for some headache sufferers. Simple relaxation methods as taught by physiotherapists may be as useful as more the more costly and time consuming technique of bio feed-back.

Drugs

- Prophylactic
- Symptomatic

Symptomatic treatment should be started as early as possible. If aura is recognized, medication should be started immediately. Once attack is fully developed, parenteral drugs are preferable over oral preparations because of decreased gastrointestinal motility and poor absorption. Aspirin, accetaminophen, propoxphane hydrochloride naproxen, ibuprofen or analgesic with caffeine may be effective. Induction of sleep, control of nausea and simple analgesics should be more widely used. 2 mg or ergotamine tartarate with simple oral analgesic caffeine combination can be repeated in 1 hour in acute attack. Metoclopromide 10mg/ chlorpromazine (25-100mg) or prochlorperazine (25mg) can be helpful if nausea is troublesome. Analgesics can be used in the form of suppository.

As an alternative to ergotamine (which should not be continuously used and avoided in hypertension, pregnancy or peptic ulcer disease to prevent ergotism) isometheptene mucate, phenylpropanolamine and pseudoephedrine, can be tried.

Breathing in and out of a paper bag to raise the PCO2 was at one time advocated to abort the aura and the following headache. Vigorous massage of the superficial temporal arteries during the aura is claimed to abort the headache.

Status migrainosis- (migraine lasts for many days with little or no relief). DHE (dihydroergotamine mesylate i.v as a bolus dose late

S.C. every 6 hours), corticosteriods by injection until vomiting ceases and then by month on a tapering dose prednisolone (20 mg 6 hourly) followed by a rapidly tapering dose over 2-3 days. Meperidine may also be needed before the above drugs take effect. New drug Sumatriptan (selective 5-hydroxy tryptamine: 5-HT, agoinst) acting on central rather peripheral blood vessels is "gold standard" for relieving moderate to severe migraine attacks, works faster when given as 6mg S/C injection into thigh and 2 injections at least one hour later. Orally 50mg/100mg upto maximum of 3 tablets in 24 hours. Side-effects include pain at infection site, tingling sensations dizziness, vertigo, drowsiness, transient chest pain and rise in blood pressure. When arterial spasm is severe and threatening the viability of extremities, sodium nitroprusside I.V should be given immediately

Prophyaxis

When the attacks of migraine occur weekly or several times a month or are very prolonged and debilitating, prophylactic medication should be started. Beta-blockers like propranolol (800-240mg/day) for 2-3 months, atenolol and metoprolol, tricyclic antidepressants-amitriptyline (10-150mg at night) imipramine and deripromine, mono-amine oxidase inhibitors-phenalzine, Cyprohepatadine is more effective in children. Calcium channel blockers like flunarizine and nimodipine and verapamil (80-120mg TDS) in classic migraine are also helpful. Aspirin taken regularly has also been advocated for migraine prophylaxis. Small doses of Sumatriptan can also be used for this purpose.

Intractable migraine

Methysergide (most intractable M) for 3-4 months at a time and should be discontinued for 2-4 weeks in between (Beware of very serious side effects). Clonidine 1.2-2.1 ug/kg, Naproxan 550 mg BD, pizotefin 0.5mg TDS or 1.5 mg HS and sumatriptan, single dose of dexamethasone combined with parentaral meperidine and promazine cocktail, are also useful.

Conclusion

Understanding the pathophysiology of the disease and avoiding the all possible triggers besides medicines, can go a long way in controlling this commonly recurring neurological disorder.

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Students Page:

A) Tips on Critical Care Medicine

Ahmed Yousf*

JK Practitioner 1996; 3:4 276-277

1. Central venous pressure

CVP - is synonymomous with right atrium pressure and is a complex phenomenon having a number of determinants. Its value reflects the state of balance between venous return to the heart, venous tone and heart functions. Variations also occur with changes in intrathoracic pressure and intra-perricardial pressure.

- It increases when venous return and venous tone increase or when cardiac function deteriorates and vice versa.

Measurement

A radio opaque catheter is passed into the right atrium from an antecubital, subclavian or internal jugular vein. The femonal veins are unsuitable because of the hight risk of sepsis.

After insertion, its position must be checked on a portable chest x-ray because not infrequently catheters double back on themselves. Pass into the vein of the neck or enter the right ventricle.

With the catheter in a suitable position, the CVP can be measured intermittently using a simple fluid manometer containing dextrose or saline or continuously with a transducer.

A fluid manometer is a branch of a given set, linked to the venous catheter via a three-way tap mounted on a centimeter scale.

Normal Values

Measured from the mid-axillary line, the normal range of CVP is 5-10 cm H_2O, from the sternal angle it is 0-5 cm H_2O.

Intermittent Positive pressure ventilator (I.P.P.V) increases The Mean Intrathoracic pressure upto 5mm. Hg. Which is reflected by a rise in C.V.P. This must be taken in account when interpret, the CVP of ventilated patients.

Clinical Application

From theoretical considerations it would appear that the CVP be used to asses blood volume (equated with venous return) right ventricular functions and by inference from this, left ventricular function.

A early accurate estimate of CVP can be made without any equipment by simply noting the height to which the external jugular vein is distended when the subject lies with the head slightly above the heart.

The vertical distance between the right atrium and the place the vein collapses (the place where the pressure is zero in it) is the venous pressure in mm.of blood.

2. Method of Arterial Blood Sampling

A-20-25 gauge needle or an indwelling needle may be used. The needle must be very sharp with a smooth level.

A shorter needle for radial puncture is generally easier to handle to avoid perforation of the posterior of the artery. The anticoagulant preferably sodium heparinate in concentration of 1000 I.U./ml heparin is drawn into syringe and allowed to coat the walls by moving the plunger back and forth.

The color of the sample is not always a good indicator of arterial blood. Patients with chronic lung village and decrease PO_2 will have darker coloured arterial blood than normal.

Technique of Arterial Puncture

The commonest sites for taking a sample of arterial blood are the radial artery at the wrist, the brachial artery in ante cubital fossa and the formal artery in the groin. In each situation the course of the artery must be defined by placing the left middle and index fingers along the line of maximal pulsation.

The needle is introduced between the two fingers and advanced slowly.

Arterial blood enters the heparinzed syringe easily and should pulsate.

After taking the sample, all air bubbles must be removed and then the end of the syringe can be sealed.

The syringe must be gently rolled between the hands to ensure that the sample is probably mixed with the heparin.

If there is any delay before analysis, the sample must be kept in ice.

Firm pressure must be applied over the puncture site for at least five minutes, otherwise, large

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Chief, I.C.U
Riyadh Central HospitalSaudi Arabia

and unsightly haematoma may form.

3. Intermittent Positive Pressure Ventilation (I.P.P.V.)

Indications

The object for I.P.P.V is to provide adequate ventilation (elimination of carbon dioxide) and oxygenation with minimal damage to the lungs and minimal disturbances of the circulation.

It is achieved by intermittently inflating the lungs with gas under a positive pressure. The natural elastic properties of under a positive pressure. The natural elastic properties of the lungs provide the necessary recoil to expel the gases.

Any system for IPPV consists of a ventilator, inspiratory and expiratory connecting tube and a humidifier since all gases given to intubated patient must be humidified. The necessary pattern of ventilation is obtained by reguting the tidal volume, the respiratory frequency (rate), the length of inspiration and expiration, and the oxygen concentration of the inspirated air. It varies from patient to patient depending of the cause and severity of the respiratory failure. usually there is no single criterion or rule that indicates the necessity for IPPV. Hypoxia, hypercanpia, excessive secretions, fatigue and disorders in the other systems often coexist and additively contribute to the need for artificial ventilation.

Endotracheal Intubation

This is necessary before IPPV can be started. An endotracheal tube of appropriate size is passed into trachea through either the mouth or nose.

Starting I.P.P.V

Typical starting values for an adult are tidal volume 12-15 ml/Kg body weight, respiratory frequency 10-14 per minute and on inspiratory/expiratory ratio of 1:2.

In hypoxia failure the inspired oxygen concentration is increased as required.

After a period of ventilation with the initial settings, gasses and any necessary adjustable are made.

In early states of ventilation, patients are often unable to adjust to the ventilator. They are restless, struggle and breathe out of phase, this reduces the efficiency of ventilation increases the risk of displaying the tube and also the likelihood of damage to the larynx. It is therefore, absolutely essential that the patient is rendered comfortable, quite and co-operative. In most severe cases the patient may have to be paralysed with neuromuscular blocking drug, curare or poncuronium.

Benefits of I.P.P.V

It supports ventilation, improves ventilation/perfusion abnormalities by opening alveoli previously closed and allows the inspired oxygen concentrations to be accurately controlled. Exhausted patients are unable to rest and sedetion can be given without fear of depressing natural respiration. The tracheal tube enables effective removal of any excess secretion.

Problems of I.P.P.V

Mechanical Problems:- Stem from obstruction and displacement of the tracheal tube and leaks or breaks in the ventilator circuit.

Circulatory Problems:- The increase in intrathoracic pressure produced by I.P.P.V initially reduces venous return to the heart, cardiac output falls and the patient becomeshypotensive. The effect is usually short lived because physiological compensation occurs, producing a higher venous filling pressure to the heart.

Infection:- Ventilators and humidifier are potential sites of bacteri colonization and present hazards of auto and cross infection.

Oxygen Toxicity:

Pneumothorax:- The positive air way pressure may rupture a week part of lung, causing a pneumothorax. This is always a serious complication and facilities for inserting a chest drain should be readily at hand.

Weaning from Respiratory Support

In general, once the respiratory failure has improved to a point where total ventilatory support is judge to be necessary, weaning can be commenced.

1. Weaning from the ventilator
2. Weaning from the tracheal intubation
3. Weaning from additional oxygen.

The whole weaning process requires careful and detailed supervisions and can last from a few hours to several days, weeks or even months.

Once the patient is able to breath sufficiently well without support and for sufficient time, the endotracheal tube can be removed.

B) Tips on Advances in Hepatology

JK Practitioner 1996; 3:4 278

1. Recent advances in Hepatology

Viral hepatitis
    New viruses identified
    New antiviral agents
    Improved methods of screening and prevention

Liver transplantation
    Auxiliary liver transplantation in acute liver failure and some inherited metabolic diseases of the liver
    New immunosuppressive agents: licensing of tacrolimus

Acute liver failure
    New definition
    Improvements in supportive care, including use of intravenous N-acetyl cysteine
    Progress in development of artificial liver

Management of bleeding varices
    Banding ligation octreotide infusions, and transiugular intrahepatic portosystemic shunting

2. Factors predicting response to treatment with interferon alfa in chronic hepatitis

Predictors of long term response
    Duration of illiness <60 years
    Age <45 years
    Total interferon dose >600 mU
    Genotypes 2 and 3
    Low level of viraemia (<350 000 hepatitis C virus RNA eq/ml)

Predictors of non-rresponse
    Body weight >70kg
    Cirrhosis
    Genotype 1
    Total interferon dose <250mU

3. Prevention and treatment of bleeding of oesophageal varices

Primary prophylaxis: blockers
Secondary prophylaxis: blockers, ? nitrates
Treatment of acute bleeding:
    Drugs: vasopressin, terlipressin, octreotide
    Entoscopic injection sclerotherapy: ethanolamine, thrombin, hystoacryl
    Endoscopic banding ligation
    Sengstaken-Blakemore tube
    Radiological : transjugular intrahepatic portosystemic shunt (TIPS)
    Surgical: oesophageal transection, portosystemic shunts

Source:
McNair ANB, Tibbs CJ, Williams R: Recent advances. Hepatology BMJ 1995; 311:1351-1355

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Am. J. of Dis. Chil., 1976, 130, P. 965-970

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NORLOX

(Norfloxacin 400mg Tablet)
The Only Alternative

Journal Scan:

Early eradicated of Helicobacter pylori is cost effective

Data from randomized, double blind, controlled trials which included patients of endoscopically proven uncomplicated duodenal ulcers is analysed. Three modes of treatments are compared; Healing with immediate eradication of H.pylori with omerprazole and amoxicillin or triple therapy (ranitidine, followed by bismuth subsalicylate, metronidazole and tetracycline or amoxicillin); ranitidine for healing and then waiting for recurrences (recurrences were treated with ranitidine, omeprazole or eradication with omeprazole and amoxicillin or triple therapy) and ranitidine for healing, maintance and recurrences. Healing with immediate eradication of H. pylori with either triple therapy or omeprazole and amoxicillin did not only have fewer recurrences but is more cost effective than eradication of recurrences or maintance with ranitidine. Drug costs were taken from Canadian national data sources and therapy costs were based on provincial data. (1993 canadian dollars)

Early eradication of Helicobacter pylori is cost effective (abstract) ACPJ club 1996; 124:53. Abstract of: O'Brien, Goeree R, Mohamed AH, Hunt R; cost effectiveness of Helicobacter pylori eradication for long term management of duodenal ulcer in Canada.. Arch. Intern Med. 1995; 155:1958-64

Cyclosporine plus methotrexate is superior to methotrexate alone for therapy in severe rhumatoid arthritis

Results of 6 months randomized double blind, placeo controlled trial involving patients of severe rheumatoid arthritis from two centres in the US and one centre in Canada (148 patients in all) in their response to cyclosporine plus methotrexate and methotrexate alone is available. Methotrexite plus cyclosporine for six months led to more patients meeting the American College of Rheumatology Criteria for improvement than did methotrexate plus placebo. (48% Vs. 16% $p<0.001$)

Cyclosporine plus methotrexate was superior to methotrexate alone for rheumatoid arthritis (abstract) ACPJ club 1996 Jan-Feb; 124:4. Abstract of Tugwell P, Pinau ST, Yocum D, et al. Combination therapy with cyclosporine and methotrexate in severe rheumatoid arthritis. N Engl J Med 1995: 333:137-41

Fibrocalculous Pancreatic Diabetes

Fibrocalculous pancreatic diabetes (FCPD) is a form of diabetes which is secondary to nonalcoholic calcific pancreatitis. It has been reported from several tropical countries. Zargar et al report its incidence from kashmir valley. 20 patients (2.55%) out of a total of 785 diabetes patients admitted to the endocrinology department of the institute of medical sciences at Soura, Srinagar, kashmir, over a period of four years, were diagnosed (WHO study group criteria) to have FCPD. The majority of these patients required insulin for the control of their blood sugar and none had consumed cassava-widely implicated in the pathogenesis of FCPD.

Zargar AH., Laway BA Masoodi SR, Shahd NA, Shahd JA,. Fibrocalculous pancreatic diabetes from the kashmir valley. Ann Saudi med 1996;16:144-147.

CASE REPORT of Myocardial Stunning from Department of Medicine, Nassau County Medical Center NY.

Myocardial stunning is defined as a prolonged myocardial dysfunction with gradual return of contractile activity after a brief episode of severe ischemia, often seen in patients of MI, angina and in those recovering from cardiopulmonary bypass. An interesting case of myocardial stunning following respiratory arrest is presented.

Riyaz B, Paddar FA, Khan FA: Myocardial stunning following respiratory arrest. CHEST; 1995; 108:1459-60.

MODERN ENDOSCOPY CENTER, JEHLUM MARKET, BATAMALOO, SRINAGAR.

The New Undergraduate Medical Curriculum for the University of Wales College of Medicine

M. Afzal Mir*

JK Practitioner 1996; 3:4 280-282

INTRODUCTION

The Education Committee of the General Medical Council is charged by statute with responsibility for promoting high standards of medical education. In their deliberations the Committee recognized the multiple problems which the current curriculum has accumulated over the last 200 years; there is emphasis on the passive acquisition of knowledge, much of it has become outdated, there is an increasing number of new specialities which over burden the curriculum, and the whole system of medical education is calculated to obstruct the acquisition of sound knowledge and is heavily in favour of the crammer and the grinder. In short, the existing curriculum which has served us well for the last 200 years had simply become overgrown and bushy. The students and teachers had difficulty seeing the wood for the trees!

THE NEW CURRICULUM

The Committee made wide ranging recommendations. Embodied in the document "Tomorrows Doctors" (1) and in common with all UK medical schools, our College has undertaken a thorough review of its medical degree scheme during the last few years, with the intention of improving its structure, aims and objectives. Whereas the focus of medical education during the present century has been mainly on the understanding of disease processes as they affect individuals, the diagnosis and management, there is a reawakening of the previous concepts in the health populations, the epidemic and environmental hazards that affect them and the means whereby diseases may be controlled or prevented. The doctors of tomorrow will also have to encounter yet unforeseen moral and ethical issues arising from the scientific advances that are to be anticipated.

In the revised curriculum, we have to distinguish the **core curriculum** where the emphasis is on the acquisition of a range of practical skills which every graduate must posses in preparation for House Officer responsibility. It would be important to demonstrate these in some form of assessment where the students would be required to show proficiency in core skills. As every student cannot learn everything and yet retain a fair degree of expertise, we have to consider that some skills may be regarded as being **optional**, which some graduates may take as additional skills whereas others may choose some other options. The Education Committee did not specify which should be the core and which should be the optional skills but they hoped that there would be a great degree of consensus on core content and required attainment at graduation in most schools.

SPECIAL STUDY MODULES (SSM)

In the new curriculum there will be greater emphasis on self-directed learning in which students do special study modules on some subjects and explore these in depth. The assessment will constitute a presentation in a verbal form and also in the form of a short thesis. The modules offered will depend on the interests, resources and individual enthusiasm of the medical school staff and also on the wider range of opportunities within the University. These papers will be laboratory based, clinical, biological or related to behavioral, research and environmental subjects.

GOALS OF UNDERGRADUATE MEDICAL EDUCATION

The Committee has put forward three goals of undergraduate medical education which should be encompassed in the new curriculum.

a) the students should acquire a **KNOWLEDGE** and **UNDERSTANDING** of health and its promotion, and of disease, its prevention and management, in the context of the whole individual and his or her place in the family & in society.

b) the student should acquire and become proficient in basic clinical **SKILLS** such as the ability to obtain a patients history, to undertake a comprehensive physical and mental state examination and interpret the findings, and to demonstrate competence in the performance of a limited number of basic technical procedures.

---

*Senior consultant Medicine &
The New Curriculum panel member
University of Wales College of Medicine
Cardiff CF4 4XN*

Received July 1996 Accepted Aug. 1996

c) the student should acquire and demonstrate **ATTITUDES** necessary for the achievement of high standard of medical practice, both in relation to the provision of care of individuals and populations and to his or her own personal development.

CORE STRUCTURE

The review of the medical cores means that traditional preclinical/clinical divide will be removed. There are two immediate effects

1. Students will have direct patient contact from year 1 in order to introduce them to clinical skills from the outset.
2. A science continuum will extend throughout the 5 years course embracing the basic medical sciences and the clinical sciences.

The new course will have two main elements.

1. A core course, representing 60 to 70% of the overall curriculum.
2. Special study modules (SSM) providing an extension to the core and furthering the range of experiences encountered by students.

The Core Course (to be followed by all students) will provide instruction on specific key skills (clinical, communication, numeracy and scientific), the acquisition of the nature of medicine. There will also be a strong element of choice of the course provided by the Special Study Modules. Project work will form the main element of SSM's with the elective period of study being a good example of such a module. Both the Core Course and SSM's will focus on a number of themes in the curriculum and will be delivered through modules or a subject panel.

THEMES

The new curriculum will have five main themes that pervade all the 5 years of the course. These are
Molecules, cells and tissues
Systems
People and population, Clinical practice
professional skills

Early stages years 1 and 2

The purpose of the first phase of the course is to introduce all the major systems of the body and indicate how they operate in the normal and diseased states. Additionally, students have some early clinical experience and develop an appreciation of the person as an individual and the role of health care in society. From a skills stand-point, from the outset students are encouraged to undertake self-direct learning and through a range of types of pleasure to work exercise some control on the direction of their studies. The main contributions will be from the following.

1. Foundation studies: An integrated course bringing into the other aspects of cell and tissue operationally with an early introduction to measurement, critical data interpretation and the use of library services and information retrievable systems.
2. Health in Society: This module has two broad strengths; the person as an individual and matters related to families and larger groups in the population. It includes topics such as personality; impact of illness, disability and death on individual and families; effects of illness in childhood and in the elderly; moral dilemmas; public health; patterns of disease; environmental and social factors; and psychological factors and disorders.
3. Musculoskeletal systems
4. Cardiovascular-respiratory systems
5. Alimentary system
6. Endocrinology, metabolism and renal system
7. Neurosciences
8. Human development, growth and reproduction
9. Mechanism of disease and therapeutic approaches
10. Defence mechanism, clinical methods

During these 2 years the students will get exposure to patients on the wards as well as to general practice out in the community on a weekly basis. They will be introduced to the concepts of history taking, communication, basic clinical skills and some ethical issues which are commonly encountered in hospitals and community practices. The timetable devised allows students to rotate around 7 one day attachments in medicine , surgery, primary care, accident and emergency, obstetrics and gynaecology, child health and care of the elderly.

Clinical training Years 3 to 5
Foundation of Clinical Skills

This comprises of 6 week course, three and half days per week. The remaining one and half are devoted to subject panels of various basic sciences and clinical disciplines. The aims of this course are to teach the students the basics of taking a full holistic medical history (including simple assessment of mental state and social history).. and to teach them to perform a general physical examination of the patient. To achieve this, the students will be divided into groups and allocated to a specific consultant medical team in South Glamorgan. The students will also be given seminars on the various aspects of history taking and examination as is illustrated from the following six examples.

1. Acclimatisation and communication

2. Producing and taking documentation
3. General examination
4. Examination of subcutaneous swellings and skin ulcers
5. History and examination of the cardiovascular system
6. History & examination of the alimentary system

Year 3 clinical training - systems

The aim of this is to deliver clinical training in a system-orientated way. However, the delivery of this will not be possible unless students have specific clinical attachments in various firms. This can be achieved by having a 5 week attachment to a medical firm during which the focus will be on teaching of diseases related to the respiratory and endocrine systems. Then there will by another 5 week term with another medical firm where the focus will be on respiratory and cardiovascular systems, psychological aspects of diseases and renal tract. Similarly there will be two more attachments of 5 weeks each where the focus will be on other subspecialites of medicine and surgery.

Year 4 speciality rotations

These will consist of 5 week rotation each in psychiatry, including learning difficulties, inpatient and outpatients psychiatry, substance abuse and a 5-week term in child health followed by a 5-week term in obstetrics any gynaecology. 5 week rotation between cardiovascular disease and respiratory medicine and finally 5 weeks in ENT, dermatology, ophthalmology infectious disease (including genitourinary medicine and haematology). At the end of the 4th year there will be a 7-week period of diagnostic synthesis in medicine and surgery. This will be joint attachment in South Wales Hospitals whereby students will be attached jointly to medical and surgical firms for a synthesis of all their clinical skill obtained in year 3 and 4.

Year 5

In year 5 the final year rotation of elective medical care within a speciality; secondary medical care within a region, and multidisciplinary medicine within the community will be provided. There will be a final attachment in the final year, of clinical consolidation of 6 weeks in which the students will spend 50% of time in medicine and surgery; they will be attached to the firms where they will be doing the House Physician job. This is essentially a taster of what they will be required to do for 12 months after graduation and will prepare them for the tasks of their house jobs.

Special Study Modules

About 30% of the total time available will be devoted to special study modules where the students will be given some clinical, laboratory and allied medical projects which they will accomplish on their own. This will be an in-depth exploration of various topics where the student will be collecting observations and synthesizing these into some meaningful conclusions. Modules can be done concurrently with the other medical rotations and also at the end of the periods there will be 7 weeks spared for special study modules. During these periods they will also have their elective when students can visit other medical schools overseas and learn how medicine is practiced in other countries.

Assessment

As in the delivery of the course, assessment will seek to gauge whether students can apply knowledge, demonstrate understanding & show an acquisition of skills. Also, because the course has a multidisciplinary structure the emphasis in their assessments is on the ability to integrate information & experiences. There will be various forms of assessments.

Regular feedback on progress is available through in-course assessment and course assessments and these are timed to permit sufficient opportunities for remedial study. Although reduced in volume, periodic complex examination will continue. These would require students to demonstrate an understanding of the various subject panels they have followed during the year. Students will also need to show that they can perform competently some of the skills that should have been gained.

SPECIAL EXAMINATIONS

1. Primary MB (Year 2)
2. Intermittent MB (Year 4)
3. Final MB (Year 5)

CONCLUSION

It is hoped that the new curriculum will embody many of the principles and objectives set out in its predecessor, together with the emphasis and alterations caused by the new developments in technology, environmental factors on illnesses, preventive strategies, and the new methods of learing. Above all the students would be encouraged to learn and there will be less emphasis on spoon feeding as was the case in the past.

REFERENCE

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   General Medical Council, London 1993.

Meet the Professor/Consultant:

G. Nabi Mir
BSc. M. Pharm; MBA; Ph. D

JK Practitioner 1996; 3:4 283

Address: Buckin Gham, PA 18912, USA

Date of Birth: June 26, 1938, Srinagar (kashmir)

Education:

- B.Sc Biology, Kashmir University, Kashmir 1985
- B.Pharm, Punjab University, Chandigarh, India 1962
- M.Pharm, Pharmacology, Punjab University, Chandigarh, India, 1964
- Ph. D., Pharmacology - Toxicology, university of Tennessee, USA, 1970
- MBA, Management, Temple University., Pennsylvania, USA 1979

Appointments:

Started as a technical assistant (58-59) in the drug Research laboratories (NC), Drinagar Kashmir; Research pharmacologist at the institute of history and medical research at New Delhi (64-66) and spired his way as a Research associate toxicology (70-71) section head pharmacology (72-83), Associate Director pharmacology (84-87) at various prestigions research institute in the U.S.A. Is the President and CEO of the Drug Research laboratories Inc, Bukingham USA, since 1988

Drug Development

Thirty seven years of drug development involvement in areas e.g. Cardiovascular, Inflammation, Gastrointestinal and Drugs from sea (Marine Toxins). Developed 12 Ind, S (intial drug applications), and 5 NDAS (New drug application submittee to approved by us FDA (Federal Drug administration). Have managed and directed all aspects of precinical (Chemistry, Pharmaceutical Development, Pharmacology, Toxicology, and Pharmacokinetics) and clinical (phase 1, Phase 2, Phase 3 and Phase 4) drug development.

Research Interest/contributions

- Development of animal models for preclinical drug research. At present provides pre-clinical espertise in new drug development as Head DRL, INC, Buchingham, USA
- Advocated development of B recep specific calcium for control of IBS and primary dysmenorrhoea.
- Demostrated antiulcer activity of almunimum hydroxide was independent of its and meatralizing acitivity.

- Advocated the concept of pre synaptic alpha advengic stimulation
- Stressed the invovement of prostoglandus in intestinal hypersecretia
- Credited with isolation of alkaloids from bulbs of FRITIUARIA IMPERIQLIS
- Two years experience of marine phamacology. Work along the Gulf of Mexico.

Professional Societies

- American Gastroenterological Association
- American Society of Pharmacology and Experimental Therapeutics
- American Association for Advancement of Science
- American Association for Pharmaceutical Scientists
- Drug Information Association
- Society of Toxicology
- Society of Sifma XI

Publications and Patents

Seventy Scientific papers and nine patents

International Scientific Journals

- Member of Editorial board of Drug Development Research.
- Reviver for several other International Biomedical and Clinical Journals
- Member of Number of us Federal Drug Administration New Drug Review committees.

Addendum:- Pg. 202, line 21 of Meet the Professor Column in the previous issue (Vol. 3, No. 3) should read after cassettes as "Ravinder's wife Usha is a teacher, zoologist by profession. She is an embodiment of courage, fartitude & wit."

Obituary:

1. **Dr. Ghulam Qadir Sabha Kashmiri**

Dr. Ghulam Qadir died at the age of 50 years at the Tata Memorial Hospital, Bombay in September 1996. He will long be remembered for his sense of humour, humble nature and poetic dispensation. May God give peace to his soul.

Editor

2. **Dr. Pran Nath Chhuttani**

Dr. Pran Nath Chhuttani, Emeritus professor of Medicine was one of the founder of PGI Chandigarh. He was a great administrator and a medical educationist, besides being a talented physician. He died on 19th July 1996. His death was mourned throughout the country.

Editor

A TRIBUTE TO DOYEN OF MEDICINE

Dr. Pran Nath Chhuttani, Emeritus professor of Medicine, Ex. Director and one of the founders of P.G.I. Chandigarh, Chairman of the Tribune Trust & Ex-President of Assoc. of Indian Doctors to fight against Nuclear explosion, breathed his last on Friday, the 19th July 1996.

A Dashing captain in the Army Medical Corps in Damascus in 1994 during the Second World War, a renowned physician, a dynamic man, a magnetic personality, a great medical teacher and an excellent research worker whose students and their students are scattered all over the World. His contributions to the medical world are unparallaled.

He was a hard task master, meticulous to the limit, his strict adherence to punctuality and discipline was a phenomenon. His quest for efficiency, hardwork and clealiness also infused enthusiasm, confidence and work satisfaction amongst all. Having worked with him as house physician and as P.G. student (M.D. Medicine) and senior resident (1971-1975), I do not think this vaccum would ever be filled. He was a doyen of the medical profession and and an embodiment of idealism.

Dr. Vijay Gupta
Professor Medicine

(Reproduced from Newsletter Vol. 2 No. 8 published by Medical Teackers Association Jammu).

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Communications to Editor:

Dear Dr. Malik,

Asalam-u-Alaykum.

Thank you for sending me the most recent edition of the JK Practitioner. Thanks to your efforts the journal is showing continuous improvement. However, I would strongly urge you to consider mechanisms whereby the quality of the pictures can be improved. We have already sent you a number of articles from here. We continue to encourage our staff to prepare more material.

Best wishes.

Sincerely yours.
Faroque A. Khan, M.B.
Nassau County Medical Center.
2201 Hempstead Turnpike,
East Meadow, NY 11554

Dear Dr. Malik,

Asalam-u-Alaykum.

I thank you for your kind letter of 04/07/96 and apologise for the delay in my reply due to the fact that I was out of station. Please tell me when is your annual issue being published and what sort of article you would like me to write. I highly appreciate your efforts. May Allah (S.W.T) reward you for all that you are doing

Dr. Muhammad Yahya Alvi
Medical Consultant cum
Director, Health Services IIUM,
Jalan University, 46700 Petaling
Jaya, Selangor Darul Ehsan.

Dear Dr. Malik,

Asalam-u-Alaykum.

Thank you very much for your letter and for enclosing the new issue of the JK Practitioner. Your Journal seems to improve by each issue and you deserve our congratulations for your endeavours. As promised, I am enclosing a report on the new curriculum which we have devised for our College. You may be aware that new curricula are being introduced in most of the medical schools in this country and in some other western countries. I anticipate that within the next couple of years you may have to go in the same direction and devise a new curriculum which looks forwards into the new millenium.

With best wishes for you and your Journal.

Yours sincerely
Dr. M.A. Mir,
Senior Lecturer & Consultant Physician,
University Hospital of Wales,
Health Park Cardiff, CF4 4XW.

Dear Dr. Malik.

It has been my privilege to know Dr Farooq Ashai Dr. Abdul Ahad Guroo and Dr. Mir Muhammed Maqbool., three of your recent profiles.

An episode which will always stay itched in my memory, was a summer night in the third week of July 1991, when on Dr. Guroo's invitation, I visited his home and found Dr. Ashai there. Considering the prevailing circumstances and the heavy burden these gentlemen were carrying, we still had a pleasant evening discussing topics, scientific, medical and otherwise. I could not escape the feeling that here are two different personalities, bound together by strong convictions apparently at peace with their beliefs and convictions but made terribly venerable by the unpredictable nature of the elements surrounding them.

Dr. Mir Muhammed Maqbool's nobility does not surprise me, it is ingrained in his genes. I want to pay tribute to him, by mentioning his late brother, Dr. mir Abdul Hamid, who met his untimely death on October 2, 1981 in an automobile accident in England

Future generations will have a path to follow which was traversed by people such as Guroo, Ashai and Mir.

Sincerely,
Ghulam Nabi Mir
President and Chief Executive Officer
DRL, Inc. P.O. Box 394
Buckingham PA 18912 USA

Dear Dr. Malik,

Asalam-u-Alaykum.

I must congratulate you for the magnitude of the academic interest you are still maintaining even in your private practice. I found your hard working nature unaltered. Believe me, as I reached here, I remembered you to my all Kashmiri friends. I displayed your JK Practitioner to them and obtained their sincere gratitude for you. This quarterly acts as an open window through which one can see the rest of the world. But I felt that you need an experienced deligient person to check the fulfilment of the guidance for authors. You are already putting your best in it to collect the material from the authors situated so far away. Whenever I am with you, I will be more than happy to serve it as much as I can.

Your brother.
Dr. Wafai.
Consulting Physician.
King Khalid Gen. Hospital.
Hafar-al-Batin, Eastern Province.
Kingdom of Saudi Arabia.

Enquiries from the eligible prospective NRI/Foreign candidates for admission to the MBBS Course of the College may be made from the address given below:-

Sd/-
Director-Principal

Office of the Imam Hussain (A.S) Medical College & Hospital
8/3, Poloview Srinagar Kashmir (India)

INDEX VOLUME 3

JK Practitioner Volume 3, No.1 (Jan-Mar), 1996

EDITORIALS:

-Medical Journalism in 20th century *G.M. Malik* 1
-From GIANTS to ROBOTS *M.A. Jallu* 3

Guest Article:

-Current concept on the management of Gall stones *R. Tandon* 7

Clinical Review:

-Cardiac Tymponade *P.K. Shah* 10

Leading Articles:

-Coronary Artery Bypass Grafting VS PTCA *P. Jain, U. Koul* 19
-NIMO DIPINE for Treatment of Stroke *S.A. Tanki, Y.A. Quazi, Z. Jeelani* 23

New Horizons in Surgery :

-Laparoscopic Cholecystectomy *N.A Wani, T. Fazili, U. Shah* 28
-Intra-ocular lens Implantation *S.J. Ahmed.* 31

Original Articles:

-Hearing Impairment in School childern of Kashmir (Epidemiological profile) *Z. Ahmed, S.A. Ahad* 34
-ACTIVATE clotting Time Estimation in Heparin reversal during open heart Surgery *A.G. Ahanger, Mehrajdin* 37
-Mixed germ cell Tumors of Testes *Chowdhery N.D., A.R Khan* 40

Case Reports:

-Papillon-Lefevre Syndrome *M. Quazi, S. Manzoor* 43
-Spinal Hydated cyst causing cord compression *G.R. Mir* 44
-PNEUMOENCEPHALUS following Nasal polypectomy *N.A. Bhat* 47

PRACTITIONERS COLUMN:

-AIDS (Part II) *G.H. Hajini* 49
-Haematuria, Do we manage it correctly! *G. Jeelani, Mumtaz D.* 53

CME Programme:-

-Seminar on T.B in Valley (How we face it!) *M. Yousf* 56

Students Page:

-Scenario of T.B. in SAARC Region 60

Meet the Professor:

-Prof. S.N. Ahmed Shah 61
Foundation of Medical College Kashmir *P. Chesti* 63

In Memorium:

-Farooq Ashai *G.H. Hajini, G.R. Mir* 66
-A.A. Beigh *Margoob, Shah, M. Yousf* 67

Overseas diary:

-History & Training of foreign Medical graduates in U.S.A *Farooque A. Khan* 69
Communications to the editor: 71

JK Practitioner Volume 3, No.2 (Apr-Jun), 1996

Editorials:

-Radio-Iodine and Thyrotoxicosis. *AH Zargar.* 73
-Aids Dementia. *PA Shah, A Nafee.* 74
-Current Postgraduate Curriculum- Need for Improvement. *A Rouf.* 75

Guest Article:

-Percutaneous Cardiopulmonary Support in high risk interventions. *FA Shawl.* 77

Clinical Review:

-Diet and Colorectal Cancer. *NA Wani, OJ Shah, T Fazili.* 82
-Approach to Child Health. *GM Dar.* 85

New Horizons in Non-Invasive Surgery :

-Extra Corporal Shock Wave Lithotripsy(ESWL). *MA Khan.* 88

Original Articles:

-Thyrotoxicosis, Management with Radio active Iodine (A Follow up study). *S Mushtaq, SH Khan.* 90
-Effect of Fluoride ions on the thyroid glands of Guinea Pigs. *Mahmood, GH Bhat.* 94

Case Reports:

-Massive Splenomegaly, an Unusual Presentation of Echinococosis. *SA Aziz, M Ahmed, MA Amin, GM Bhat, A Lone, G Nabi, H Bano.* 97
-Adenoma Upper Lip (Papillary -Type). *B Ahmed, M Shafi, N Rashid.* 99
-Body Favus. *Q Masood, S Manzoor.* 100
-Mexiletine Toxicity. *SB Bansal, HS Rissam, FA Bhat, J Ashraf.* 101
-Tuberculous Parotitis. *NA Bhat.* 102

Special Article:

-Organ Transplantation. *MA Albar.* 104

Practitioners Column:

-Allergy and Chronic Allergic Rhinitis. *MM Maqbool.* 109
-AIDS in Children (Part III). *GH Hajini.* 111

Students Page:

-Cosmetics in Practice. *S Manzoor, S Parveen.* 113
Clinical Images: *AH Zargar.* 116

Hospitals Today:

-Performance Appraisal A Subjective Phenomenon in Hospitals. *MA Wani.* 117

Letter to Editor:

-Heriditary Orotic-Aciduria can be caught. *ST Hussain.* 119
-Pulse Polio Campign in valley. *MA Shawl.* 120

Meet the Professor:

-Prof. M. Maqbool (ENT). 121
-Prof. Upendra Kaul (Cardiology). 122

In Memorium:

-Late Prof. A. A. Guroo. *A Ahad, Ashaq Hussain.* 123

Overseas diary:

-Life as a Medical Student in th U.K. *Monica Mathur.* 124
-Issues faced by IMGS and Programme Directors in USA. *FA Khan.* 126

Communications to editor:

JK Practitioner Volume 3, No.3 (Jul-Sep), 1996

Editorials:

-Tobacco Smoking. The Health Hazard & Beyond — *Sheikh Aejaz Aziz* — 128
-Brain Death — *A. Salam, A. Wahid, M.A. Akhter* — 129

Guest Article:

-Management of Chronic Hepatitis — *M.P. Sharma* — 131

Clinical Review:

-Post-MI Mechanical Complications — *P.K. Shah, J.S. Goodman* — 136
-Biochemical Assessment of Hepatobiliary Disorders: Traditional Approaches with New Strategies — *M.M. Mir, A.R. Khan, Z.A. Shah* — 142

Original Articles:

-A Survey of the Value of Ultrasound as a Predictor of the Findings at ERCP in a District General Hospital — *M.R. Teli, I. Gobden* — 145
-Chemical Composition of Gall Stones — *A.M. Bhat, I.A. Mir, O.J. Shad, N.A. Wani* — 151
-Role of Colposcopy in Cervical Cancer Detection — *W. Khannam, S. Hamidi, A. Rashid* — 155

Case Reports:

-Survival in Water Intoxication — *S. Banzal, F.A. Bhat, H.S. Rissam, Jamal A.* — 157
-Carcinoma of the External Auditory Meatus — *N.A. Bhat* — 160
-Kartagener's Syndrome with Simple Hepatic Cyst — *S.A. Kadla, S.A. Khan, M. Agaz, M.A. Wani, T. Hussain, S. Rashid, J. Basu, G.M. Malik* — 162
-"Xeroderma Pigmentosa & Skin Cancers" — *S.A. Ahad, M. Latif, Zahoor A., Naseema C.* — 165
-Supraglottitis During Pregnancy — *Parveez A. Mir* — 167

Short Case Report with Clinical Review:

-Extreme Metabolic Alkalosis & its Management: A Review of Two Cases — *R. Bashir, T. Mir, F. Khan* — 169

Special Articles:

-Doctor & Patient Relationship "Holding a Hand is Often as Important as Examining One" — *Lawrence H. Brandt* — 173
-"Islamic Teachings & Cancer Prevention" Tobacco & Cancer — *M.A. Albar* — 178

Imaging Today:

-Ultrasonography in Chest Medicine — *G.Q. Khan, Iftikar A.* — 185
-Magnetic Resonance Cholangiography- a Threat to Diagnostic ERCP? — 187

Practitioners Column:

-Laboratory Diagnosis of HIV Infection — *G. H. Hajini, S.T. Hussain* — 188

Students Page:

-"The Psychology of Resuscitation" — *Ralph M. Shenefelt* — 193
Clinical Images: — *Mir N. Ahmad* — 194

Hospitals Today:

-Implementing & Sustaining Health Service Quality — *Syed Amin Tabish* — 195

Overseas diary:

-Future Prospects for International Medical Graduates in the United States — *Tanveer P. Mir, F.A. Khan* — 198

Meet the Professor:

-Prof. Brij Mohan Bhan — *M.S. Misgar* — 201
-Prof. Mohd. Aslam — 203
Obituary: — 204

Communications to Editor:

JK Practitioner Volume 3, No.4 (Oct-Dec), 1996

Editorials:		
-Sepsis Syndrome Therapy	*Farooque A Khan, R Bashir*	206
-The Chronicle of Growth Hormone Insensitivity	*AH Zargar, BA Laway* *SR Masoodi, FA Sofi, AI Wani*	209
- Postmenopasual Hormone Use-Time for Concensus	*Bilquees Jamila, N Firdous, Zaffar Abbas*	212
Guest Article:		
-Fulminant Hepatis Failure	*PK Garg, Rakesh K Tandon*	214
Clinical Reviews:		
-Application of Laser in Sugery	*NA Wani*	220
-Ischaemic Strokes:- An Update	*PA Shah*	222
Mini-Reviews:		
-Gastrointestinal Mucosal Protective Activity of Aluminium Ion-A	*GN Mir*	226
-Tobacco & the Integument	*GH Hajini, Pervaiz Shah, Iffat Hassan, Abdul Ahad*	229
Original Articles:		
-The Pattern of Child Psychiatric Disorders in Kashmir	*Mushtaq A. Margoob*	233
-Tumor Pattern in Undescended Testis	*ND Choudry, AR Khan*	237
-A Study of Trace Elements, Copper and Zinc in Patients of Head, Neck and Oesophageal Malignancies	*SJA Bihaqi, M Latif Sheikh, Abdul Ahad*	241
-Fine Needle Aspiration Cytology as an Initial Tool in Evaluation of Head and Neck Masses	*MA Darzi*	245
Experimental Medicine:		
-Lithium and Kidney	*MS Shah, Hassan G*	249
Case Reports:		
-Clouston Syndrome	*Masood A Qazi, S Manzoor, Aftab Ahmad*	251
-Late Metastatis of Malignant Melanoma to Bladder	*AA Bhat*	252
-Recurrent Ameloblastic Fibroma with Atypical Presentation	*Naseer A, MA Darzi, Shabir Iqbal, ZA Khan*	253
Special Articles:		
-"Islamic Teachings & Cancer Prevention" Part II - Alcohol and Cancer	*MA Albar*	255
-Glimpses of Research in Medicine in the Valley for the Last Two Decades	*Mohd. Yousuf*	260
Imaging Today:		
-Magnetic Resonance Imaging & Proptosis	*S Sajad, Bashir A, AR Nasti, Tejit Singh*	263
-Ultrasound Pictures of GIT Ascariasis	*GM Malik, S Kadla*	265
Clinical Images:	*AH Zargar, SR Masoodi, BA Laway, S Iqbal*	266
Doctor and Patient Relationship:		
-Medical Ethics	*S Amin Tabish*	267
Practitioners Column:		
-Migraine - New Horizons	*Abdul Salam*	273
Students Page:		
-Tips on Critical Care Medicine	*A Yousif*	276
-Tips on Advances in Hepatology	*Roger William*	278
Journal Scan:	*Zaffar Abbas*	279
Overseas diary:		
-The New Undergraduate Curriculum for the University of Wales College Of Medicine	*AM Mir*	280
Meet the Professor:		
-Dr. G.N. Mir		283
Obituary:		284
Communications to Editor:		285
Index Vol. 3 (Nos. 1-4)		

Information to Authors

The JK - PRACTITIONER Journal will henceforth receive and consider for publication, manuscripts prepared in the Vancouver Style (see Lancet : Feb, 24, Page 429, 1979) and manuscripts should be addressed to:

Dr. G M Malik, Chief Editor
JK Practitioner.
P.O. Box 884
GPO Srinagar - 190 001, J&K (India)

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